

Journal OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

AVMA Convention—Philadelphia, August 18-21, 1958

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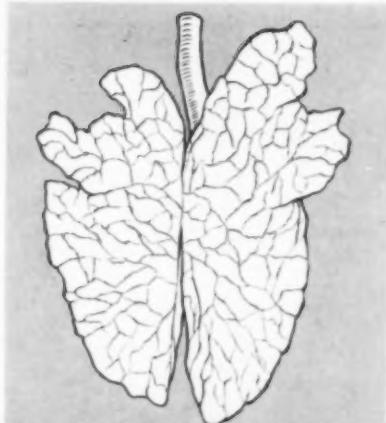
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Correspondence

June 23, 1958

Dear Dr. Aitken:

I write this about our *Veterinary Bulletin* which has a long-standing exchange with your JOURNAL of the American Veterinary Medical Association, and which provides a service to all veterinarians by abstracting the veterinary literature from all parts of the world.

My object is to seek your advice on how this journal and our other publications, i.e., *Veterinary Review* and *Annotations* and *Index Veterinarius* could best be brought to the notice of veterinarians in the United States.

Our finances unfortunately prevent us from launching out on an expensive advertising campaign. To explain why that is so, it is necessary to give you a brief account of the organization of this Bureau.

Started in 1930, this Bureau forms part of an organization known as Commonwealth Agricultural Bureaux (C.A.B.) formed with the object of collecting, collating, and disseminating information on all branches of agricultural science.

C.A.B. is financed by contributions from all the governments of the countries which make up the British Commonwealth of Nations, namely the U.K., Canada, Australia, New Zealand, South Africa, India, Pakistan, Ceylon, Ghana, the Federation of Rhodesia and Nyasaland and the smaller territories administered by the Colonial Office.

The increasing costs of printing, paper, salaries, etc., since World War II have increased the subsidy necessary.

The only method open to us to check the rising cost is to increase sales of our publications. At present, sales only meet about a third of the cost of production.

Although we have a number of subscribers in your country, I feel that there is scope for considerable expansion if our journals could be brought to the notice of a larger number of American veterinarians.

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For that purpose there is no better medium than your journal and I would enquire whether you would be agreeable to some system of exchange advertisements between our journals, or can you perhaps suggest some alternative method?

As a nonprofit organization which renders a worldwide service to veterinarians, I feel confident that our suggestion will receive your sympathetic consideration.

Yours sincerely,

M. CRAWFORD,
Commonwealth Bureau of Animal Health,
New Haw, Weybridge, Surrey, England.

[We are pleased to publish the above letter since it indicates how information regarding veterinary medicine is disseminated on a world-wide basis. The *Veterinary Bulletin*, a monthly publication, presents concise abstracts on about 4,000 articles annually. All who are doing research on veterinary medical or associated subjects should have access to this unique publication. Subscription price 70 s. (about \$10).—ED.]

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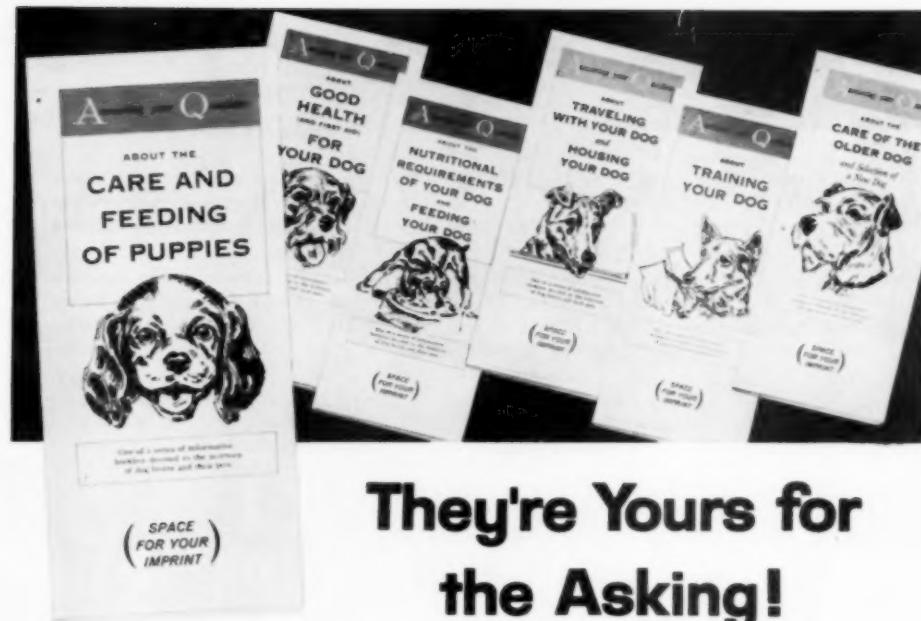
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AVMA Report

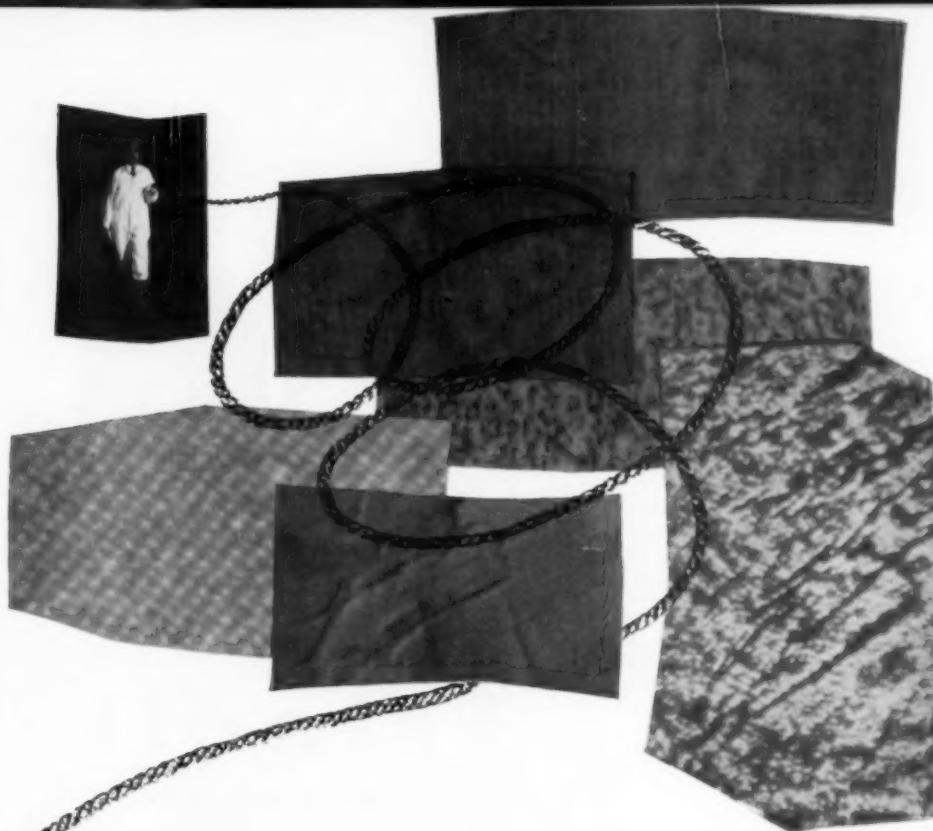
Last Call for Philadelphia 95th Annual AVMA Meeting —August 18-21, 1958

There are many good reasons for a member to attend his national association convention . . . to hear reports from top veterinarians . . . to discuss animal disease problems with colleagues . . . to renew old friendships . . . or just to take a break from daily routine to "get away from it all."

One more very good reason this year is the opportunity for you to see the new AVMA exhibit, "AVMA IN ACTION," developed so that all veterinarians may better understand how the AVMA functions in their behalf. You will learn how you, as an individual, benefit from organized veterinary medicine, told in a most interesting manner by this new exhibit.

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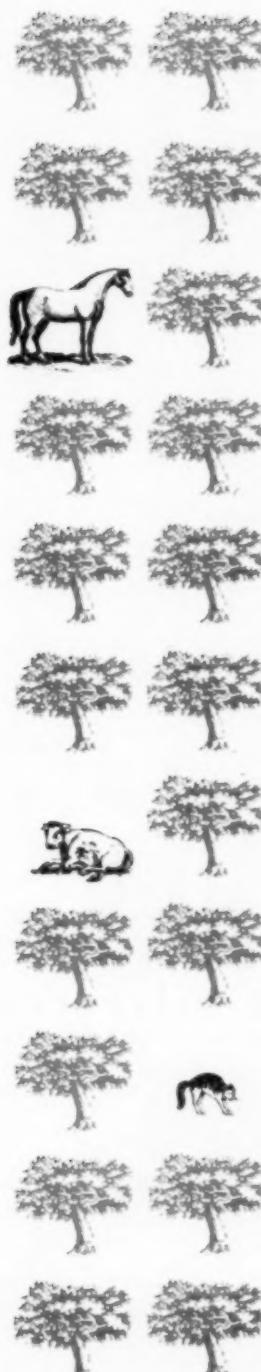
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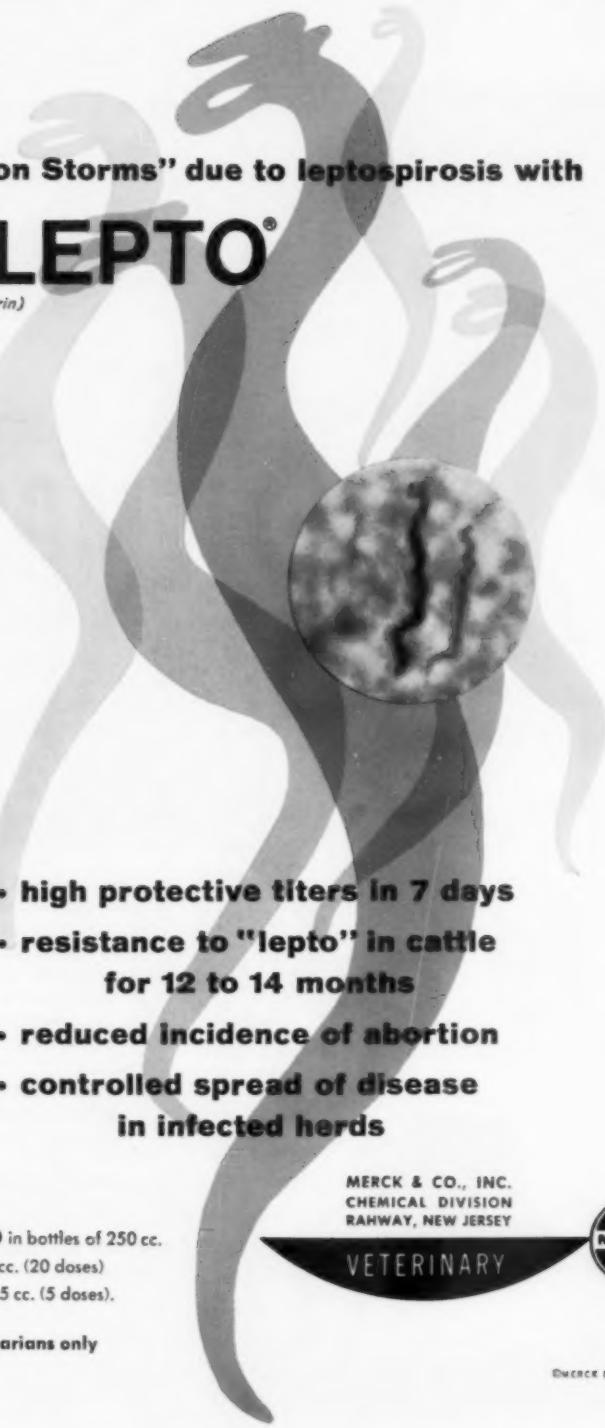
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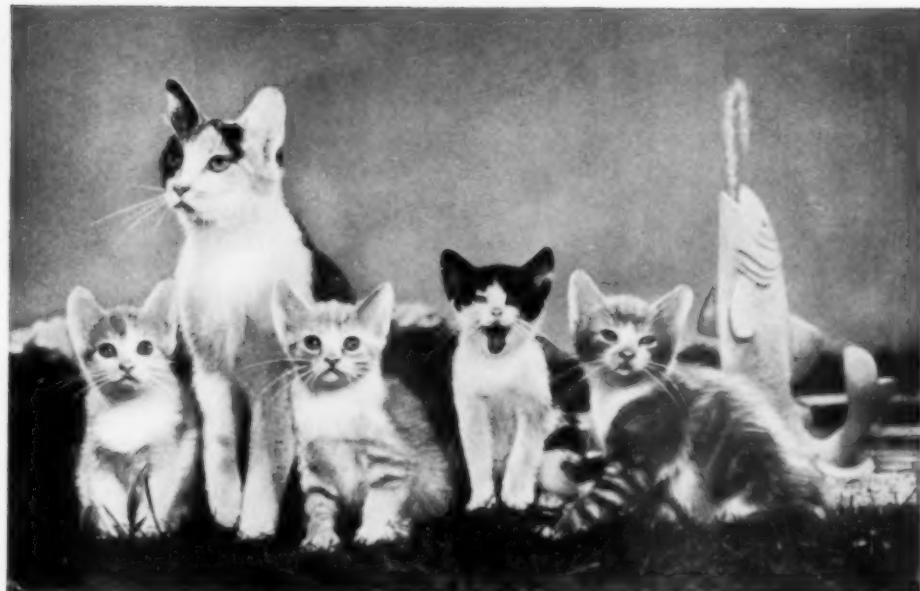
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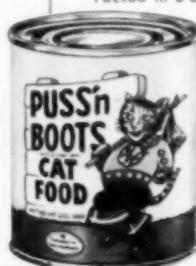
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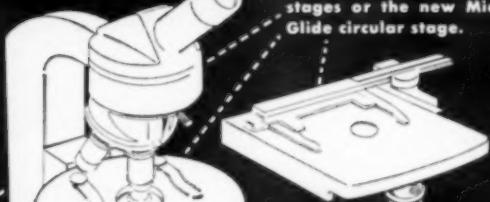
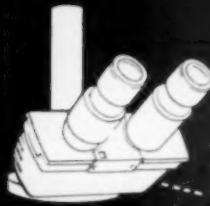


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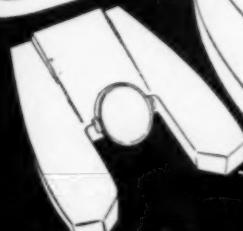
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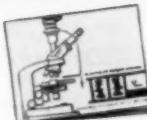


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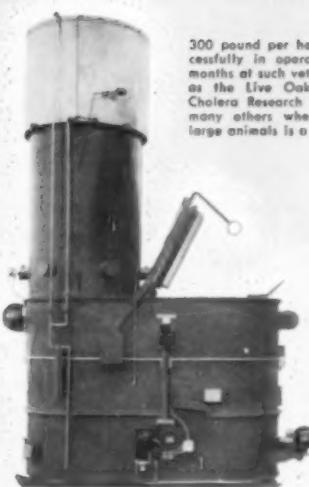
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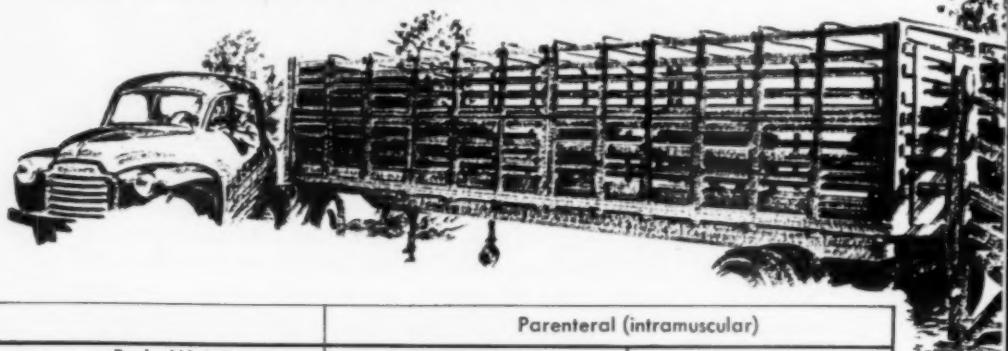
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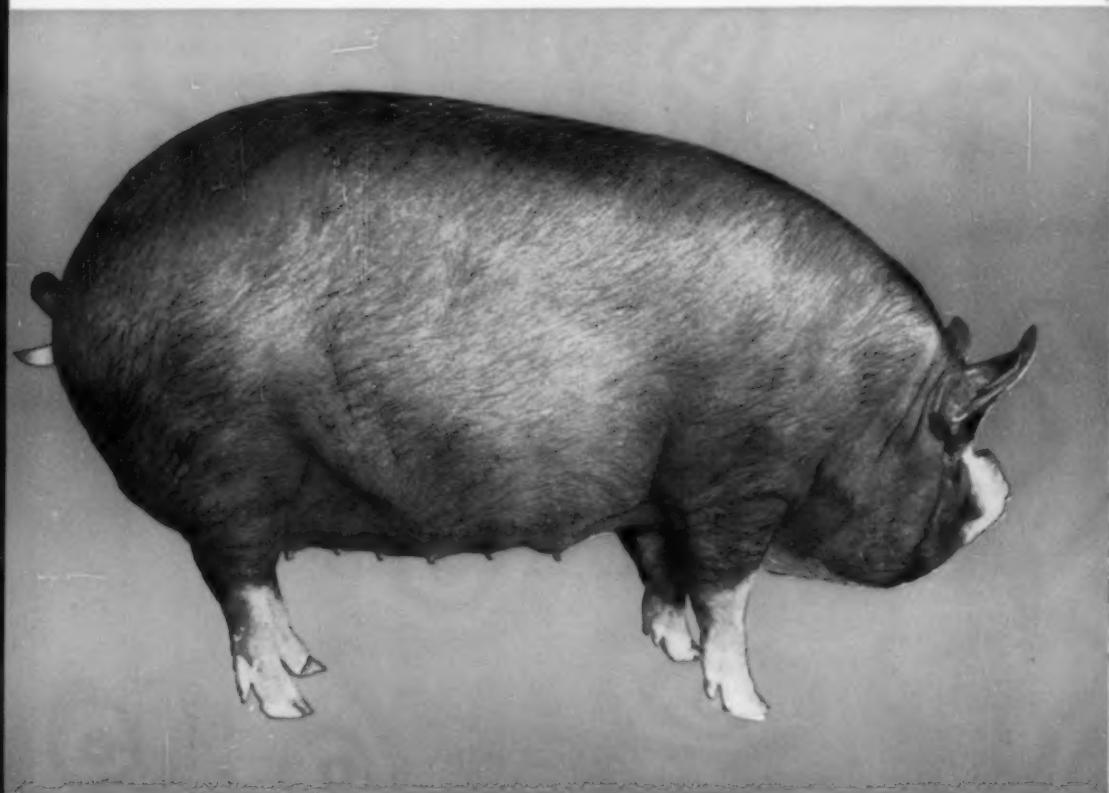


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Radiographic Anatomy of the Canine Skull

W. C. D. HARE, B.Sc., Ph.D., M.R.C.V.S.

Guelph, Ontario

THIS PAPER is presented in the hope that it will help the practitioner with his interpretation of radiographs of the canine skull.

of skull, respectively. It should be borne in mind, however, that the illustrations (fig. 1-10) can be used only as a guide because

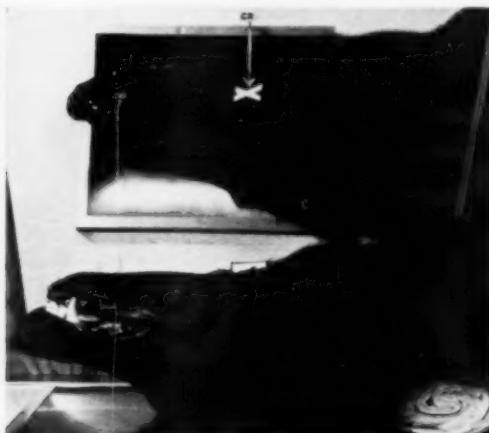


Fig. 1—Position of the dog for a ventrodorsal view of the skull. Notice that the neck is raised so that the ventral surface of the lower jaw is parallel with the film. The central ray (CR, arrow) is perpendicular to the film and directed to the body (basihyal bone) of the hyoid bone.



Fig. 2—Position of the dog for a lateral view of the skull. Notice that the face and lower jaw are raised until the sagittal plane of the head is parallel with the film. The central ray (CR, arrow) is perpendicular to the film and directed to the middle of the zygomatic arch.

A 15-month-old German Shepherd bitch and an aged English Bulldog bitch were used as subjects, since they represent the dolichocephalic and brachycephalic forms

distinct variations occur between the sexes and within the same breed.

The dogs were anesthetized with barbiturates and positioned as shown in figures 1 and 2. Parspeed screens and regular film were used without a grid at a focal-film distance of 36 inches.

See figures 3 to 10 on pages immediately following ➤

From the Ontario Veterinary College, Guelph, Canada. Dr. Hare is now at the School of Veterinary Medicine, University of Pennsylvania, Philadelphia.

The author acknowledges with gratitude the advice and encouragement received from Dr. J. H. Ballantyne, the helpful cooperation of the Department of Medicine and Surgery, and the work of the Department of Photography.



Fig. 3—Ventrodorsal view of the skull of a female German Shepherd.

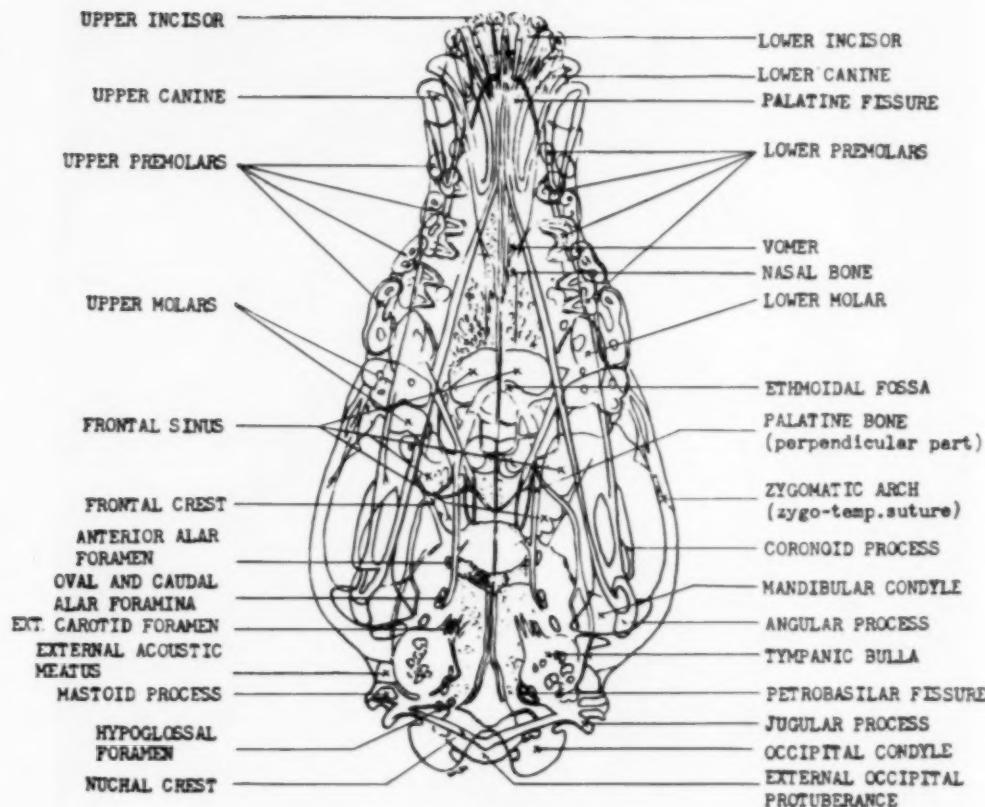


Fig. 4—Labelled tracing of figure 3.



Fig. 5—Lateral view of the skull of a female German Shepherd.

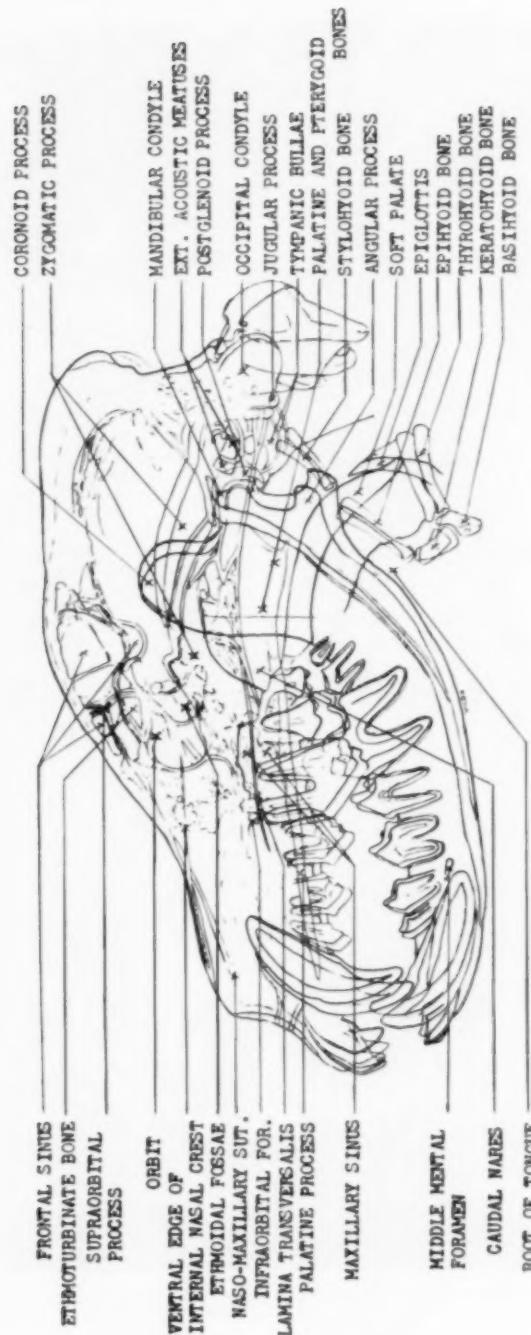


Fig. 6—Labelled tracing of figure 5.



Fig. 7—Ventrodorsal view of the skull of a female English Bulldog.

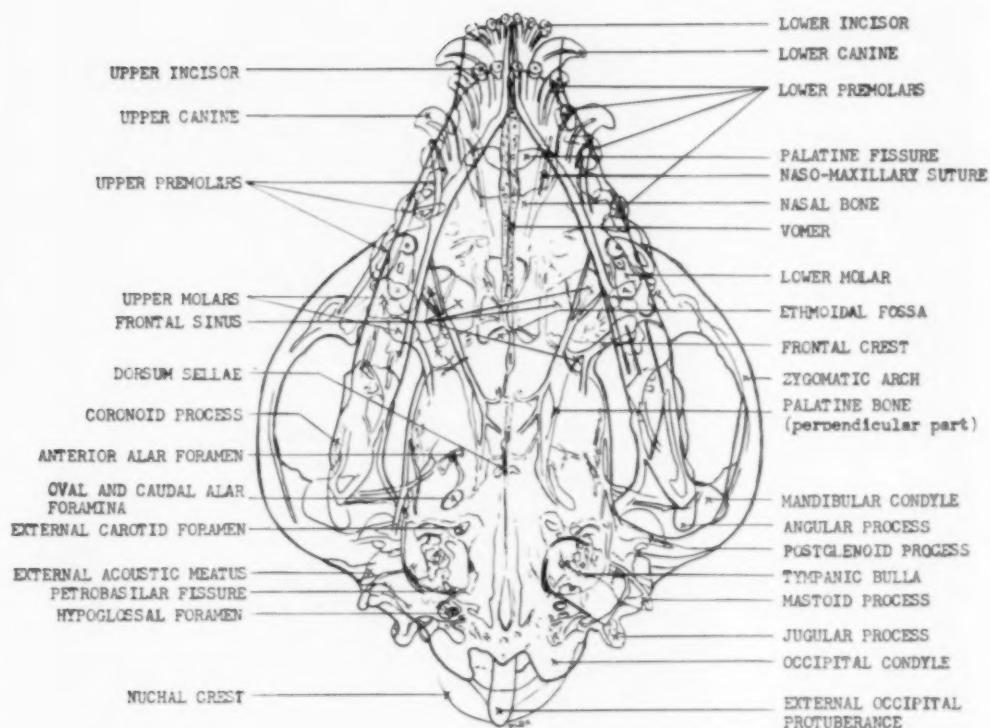


Fig. 8—Labelled tracing of figure 7.



Fig. 9.—Lateral view of the skull of a female English Bulldog.

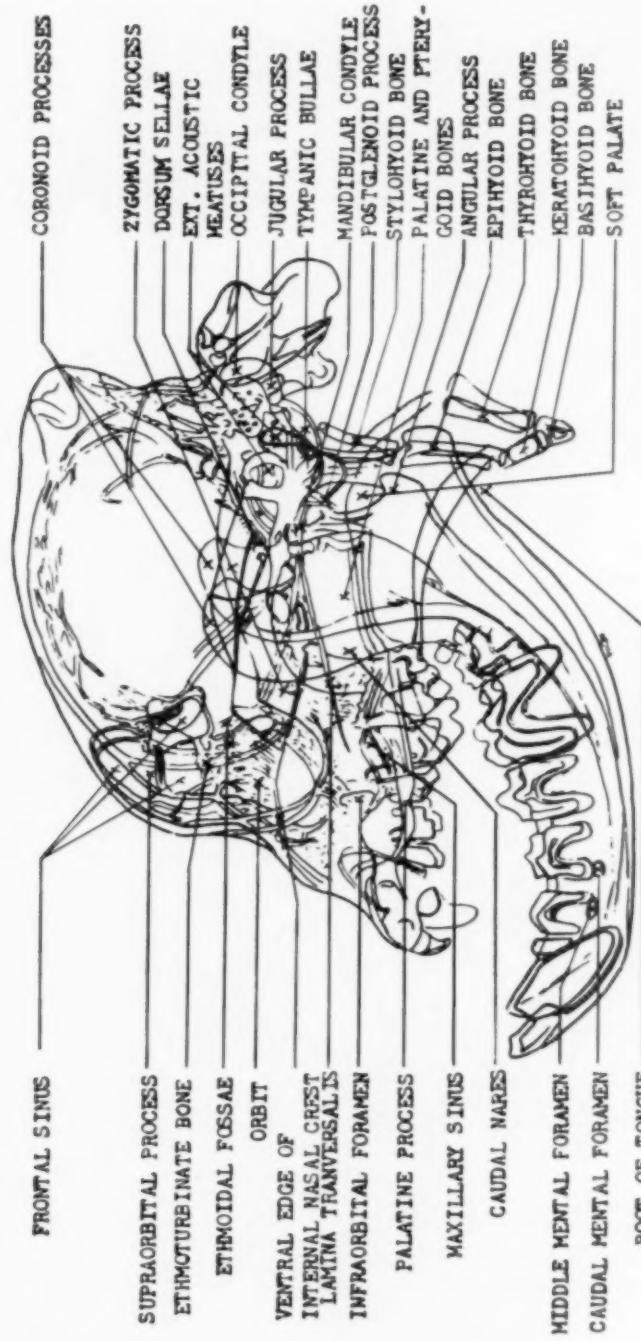


Fig. 10—Labelled tracing of figure 9.

Surgery and Obstetrics

and Problems of Breeding

A Multipurpose Wound Retractor for Small Animal Surgery

JOHN G. BOWNE, D.V.M., M.S.

Ames, Iowa

There are few surgical retractors that are designed specifically for the veterinarian; however, many wound retractors have been designed for the human surgeon. Unfortunately, these will not always serve the needs of the veterinary surgeon.

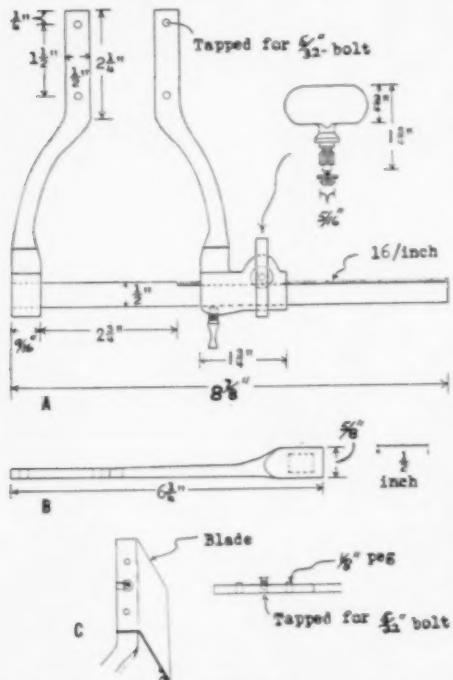


Fig. 1—A line drawing of the multipurpose surgical retractor: detailed drawing of the retractor and pinion (A); lateral drawing of the retractor arm (B); a method of attaching blades to the retractor arms without removing the bolts (C).

The inadequacy of an available wound retractor became a problem during the initial phases of a research project which entailed a hemilaminectomy of the last three cervical and first thoracic vertebrae in the dog. Several of those used for human

Dr. Bowne is assistant professor, Department of Veterinary Anatomy, Iowa State College, Ames.

The author thanks Robert Billiar for the scale drawing of the retractor.

surgery were tried but none of them provided adequate visualization of the operative field.

The distance from the dorsal aspect of the neck to the dorsal arch of the vertebrae varies with the breed; therefore, there must be replaceable blades on the retractor arms so they can be adjusted to the size of the dog. The distance between the retractor arms should be sufficient to allow both of the surgeon's hands to have free access to the depth of the wound. The retractors designed for human use were either too large and unwieldy or the blades were not of satisfactory design. The blades on the retractors for human ribs are too deep and too heavy for optimal design in small animal surgery. If they are allowed to rest on the rib cage, the excess metal protruding into the thoracic cavity soon damages the lungs during normal respiratory movements.

As the veterinary surgeon often works alone or with a minimum of semiskilled or unskilled assistants, good wound retraction is essential for the success of the operation.

RETRACTOR FOR VETERINARY SURGERY

The first model of our retractor, with its removable blades (fig. 1), was made mostly by hand. In the original model (fig. 1A), the bolts that secure the blades to the retractor arm had to be completely removed before the blades could be changed. Since this is inconvenient, if it is necessary to change retractor blades during an operation, a more convenient way to rapidly secure the various blades to the retractor arms was devised (fig. 1C).

The retractor is made entirely of stainless steel.* The retractor arms, which are fashioned from $5/8$ -inch square stainless steel bar, were bent to the desired shape in a large machinist's vise. The excess steel was removed with a coarse grinding wheel.

Various views of the retractor and some of the blades that might be used with it are shown (fig. 2). The first blade, designed for abdominal wall retraction, is curved so as to better conform to the shape of an elliptical incision. The second blade was used to retract the thick neck muscles just anterior to the spines of the thoracic vertebrae.

*The rack and pinion used in the retractor were made at the instrument shop at Iowa State College.

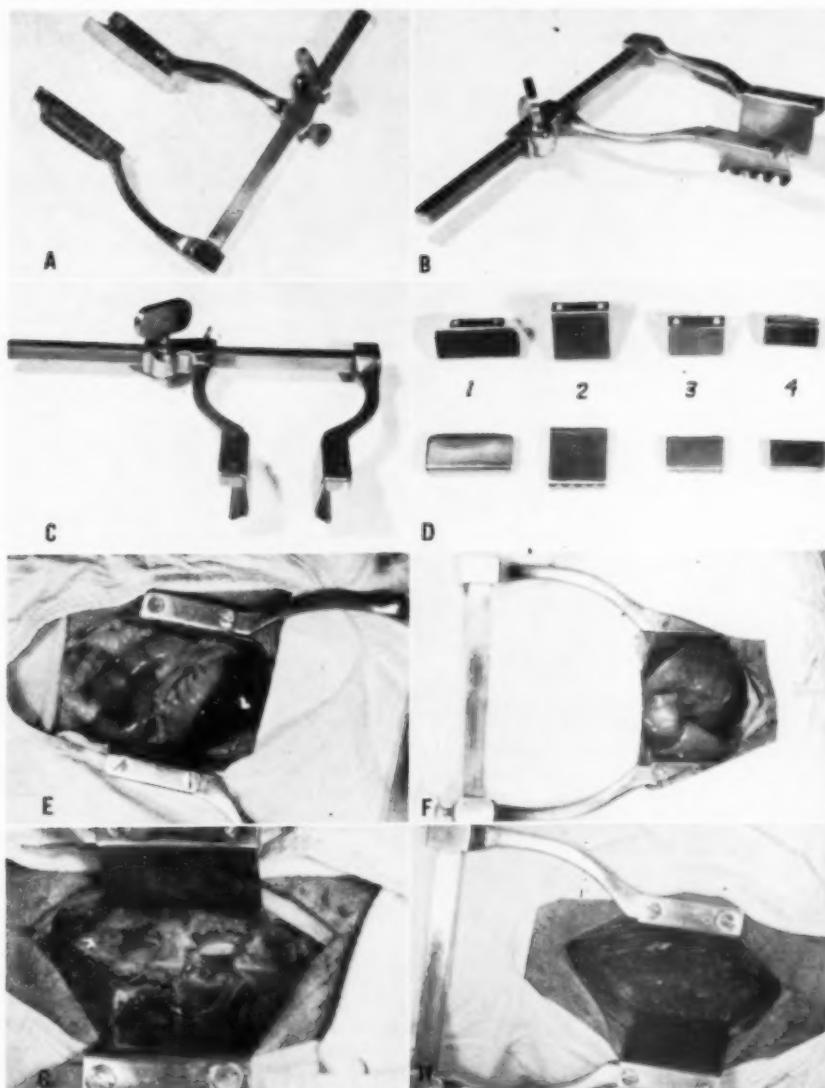


Fig. 2—Views of the multipurpose wound retractor and retractor blades: top view (A); three-quarter view (B); anterior view (C); retractor blades (D)—abdominal blade (1), deep muscle blade (2), short muscle blade (3), rib retractor blade (4); abdominal blades in use (E); rib retractor blades in use (F); muscle retractor blades in use—the short muscle blade (above) is against the spinous processes of the lumbar vertebrae, while the deep muscle blade is holding the lumbar musculature away from the vertebrae—notice the easy access to the spinal cord and vertebral canal (G); deep muscle blades in use in the thick neck muscles (H).

The use of a combination of blades 2 and 3 is advantageous in intervertebral disk surgery in the thoracolumbar area. The short blade is placed against the

spinous processes of the spine and the deeper blade is used to retract the lumbar muscles laterally (fig. 2G), since it allows a clear view of the spinal cord and the

structures which are to be manipulated, or the disks which are to be fenestrated.

The fourth blade is used in the rib retractor. The depth of this blade is gauged for the average sized dog. The rib retractor is shown in place in a large dog (fig. 2F). Nothing remains below the parietal pleura to rub against the delicate surface of the lungs.

The various blades are fashioned from heavy-gauge stainless steel sheet, approximately 1/16 inch in thickness. The muscle retractor blades have five serrations on the bottom curve (fig. 2B) to facilitate better contact with soft tissues. These serrations are smooth and should not cut or unduly damage muscle tissue. The muscle blades are set on the retractor arms so that they taper toward each other at the bottom (fig. 2B) so as to conform to the desired tapering of the wound, which is widest on the surface and narrows considerably toward the bottom.

This retractor has been used under a number of experimental as well as clinical conditions and it has been found to be as valuable as an extra pair of hands. The fact that it does not tire or obstruct the operative field makes it, at times, as valuable as an assistant.

Preparturient Temperature Variations of the Bitch

In 39 bitches investigated, the body temperature gradually decreased in four to six days to 37.0 to 37.2 C. (98.6 to 99.0 F.) 16 to 24 hours before parturition, then returned to normal at the time of parturition.

High temperatures of 39.0 C. (102.2 F.) or low temperatures of 36.6 C. (97.9 F.) before parturition seem to indicate a complicated delivery and a necessity for chemotherapeutic or surgical intervention.

A temperature of 39.0 to 39.5 C. (102.2 to 103.1 F.) on the first or second day after parturition may indicate need for an acceleration of the involution process.—*Magyar allatorv. Lap.* (Dec., 1957): 381.

Epiphysiodesis.—Epiphysiodesis is the intentional early arrest of growth at the epiphysis to reduce the longitudinal growth of a bone. Lasting arrest of growth can be effected by the surgical removal of bone at the epiphysis (a complicated procedure) and temporary arrest can be achieved by

using three strong staples across the epiphysis, one leg of each in the metaphysis, the other in the epiphysis.—*J.A.M.A.* (April 12, 1958): 1912.

Effects of Stilbestrol on Calves

Diethylstilbestrol (stilbestrol) was given to yearling heifers, heifer calves, and bull calves for approximately five months to determine any abnormalities in the reproductive organs or in sexual behavior attributable to the treatment.

Yearling heifers showed no differences in the frequency and duration of estrus or in fertility. Estrogenic assays indicated that over 40 per cent of the estrogen equivalent of stilbestrol intake was eliminated in the feces.

Stilbestrol seemed to hasten and accentuate the onset of puberty in heifer calves. However, no observable consequences, as measured by semen evaluations and comparisons of testicular size, were observed in treated bull calves.—[H. W. Reuber: *Effects of Diethylstilbestrol Feeding on the Bovine Reproductive Tract*. *Am. J. Vet. Res.*, 19, (July, 1958): 585-590.]

Effect of Estrogen on Sex Ratio.—

When 34 Brown Leghorn pullets and a few males were given stilbestrol tablets (30 mg.) intraperitoneally, there was no evidence that treatment of the males affected the sex ratio of their progeny, whereas there was a preponderance of female offspring of the treated hens. The greatest effect was at four to seven months after implantation, after which the sex ratio of the progeny reverted to normal. There was no evidence that this was due to a selective mortality of males nor could it be proved that this was due to sex reversal of males.—*Poult. Sci.* (March 1958): 307.

A Prolific Sow.—A registered, 2-year-old, Landrace sow, in Missouri, during 1957 farrowed 54 pigs and raised 42. In January, she farrowed 16 pigs and raised 12; in June, 21 and raised 17; in November, 17 and raised 13. As a yearling, she had farrowed 25 pigs and raised 24, a total of 79 pigs farrowed and 66 raised in five litters.—*Nat. Hog Farmer* (May, 1958): 14.

Problems of the Dairy Bacteriologist Resulting from Mastitis

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IMPROVING THE QUALITY of milk is a never-ending task which requires the concentrated efforts of many people with diversified interests, all of whom have a definite part to play in production of milk of high quality. The owner or manager of a dairy farm, the dairy bacteriologist, the fieldmen employed by milk plants, manufacturers of milking equipment, chemical companies manufacturing detergents and bactericides, the veterinarian, and several others must cooperate in their efforts to attain quality milk.

The dairy industry desires to sell milk of good quality at the lowest possible cost to the consumer. A poor quality raw product increases the cost of milk because of the additional time and attention that the fieldman must give to the producer of such a product. More than the usual number of laboratory tests must be conducted on such milk to determine whether it meets the standards of a particular city.

One of the methods used for assessing the bacteriological quality of milk is the microscopic method. In this method, 0.01 ml. of milk is spread over 1 sq. cm. of a glass slide and permitted to dry. The dried smear is then defatted, fixed, stained, and examined under the microscope, using a magnification of about 1,000 times. This method permits counting of bacteria, blood cells, and epithelial cells.

Those who have examined samples of milk by the microscopic method over a period of years have noticed an increase in the numbers of leukocytes and epithelial cells. This indicates that not only is the problem of mastitis in dairy herds unsolved, but it is perhaps worse today than it was several years ago.

There are several reasons for an increase in the number of cows with mastitis on dairy farms. Before antibiotics were available, a cow with a severe case of masti-

tis probably would have been sold for slaughter and thus removed from the dairy herd. Today, with the large number of antibiotics available for treating cows with mastitis, herdsmen are inclined to keep such animals in the herd, hoping that one treatment or another will cure the animal. The milk from such cows probably is included in the regular milk supply, thus accounting for the high leukocyte counts.

Most of the major cities in the United States have adopted the "Milk Ordinance and Code" recommended by the U.S. Public Health Service. This ordinance defines milk as "—the lacteal secretion, practically free of colostrum, obtained by the complete milking of one or more healthy cows, which contains not less than 8.25 per cent milk solids-not-fat and not less than 3.25 per cent milk fat."

According to this definition, the lacteal secretion from cows having mastitis is not milk. Cows having mastitis are not healthy cows and, in addition, milk from them may not meet the standards for solids-not-fat and milkfat. The code states that "cows which show a complete induration of one quarter or extensive induration of one or more quarters of the udder upon physical examination, whether secreting abnormal milk or not, shall be permanently excluded from the milking herd: provided, that this shall not apply in the case of a quarter that is completely dry."

If all of the cities which have enacted the "Milk Ordinance and Code" would enforce all provisions of the ordinance, perhaps more improvement would be made in the control of mastitis. These cities have the necessary legislation to eliminate milk from cows with mastitis from their supply, but it is not being done. If all milk from mastitic cows were rejected, we would not have a surplus of any dairy product, but a shortage. A lack of the necessary personnel is perhaps one reason why this milk is not rejected; lack of satisfactory testing procedures is another.

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DETECTION OF MASTITIS

Many methods have been suggested for the detection of mastitis. Some are based on changes in the composition of milk (lactose, chlorides, solids), the pH, the cell count; others on the organisms involved. When many methods are suggested to attain a certain result, it generally indicates that all of the methods have some limitation. This seems to be the case in the detection of mastitis. No one method is completely reliable and, consequently, several methods are employed simultaneously.

As early as 1918, it was stated¹ that if a maximum cell count could be established as a point at which milk might be said to come from diseased udders, it would be of great value. Numerous investigators have reported what they determined to be a normal leukocyte count for milk. Some leukocytes are found in the milk of any cow; counts of 43,000 to 900,000 per milliliter, in normal milk, have been reported.

Such factors as variations in the stage of lactation and method of sampling may account for such wide variations in the leukocyte count of normal milk. The results from one study² can be summarized as follows:

1) The cell counts of milk from healthy cows may be expected to be over 1,000,000 cells/ml. for the first two days after parturition, but from then on there will be a gradual decrease in numbers until the third or fourth week.

2) By the middle of the third week after calving, the cell count of milk from healthy cows may be expected to have reached almost a normal level—approximately 70,000 cells/ml.

3) The geometric average cell count of milk from healthy cows, from the second through the forty-first week after calving, is approximately 70,000 cells/ml.

4) The weekly mean cell counts of milk from a healthy cow may differ significantly from week to week. Also, differences in cell counts of milk occur among healthy cows in the same stage of lactation.

From the standpoint of reliability, leukocyte counts are considered the most accurate method of detecting mastitis. The procedure is not adaptable to routine use by a milk producer on the farm; it is a laboratory procedure which requires a trained technician.

Certain practices will increase the leukocyte count of milk: the injection of distilled water or physiological saline solution into the udder; too vigorous manipulation of the udder, e.g., from leaving a milking machine on too long; and the cessation of, or intermittent and incomplete, milking. The latter practice not only gives a high cell count but also an increase in the number of bacteria in the milk. Micrococci are present in the udders of healthy animals, a fact not recognized by some individuals.

If the leukocyte count is to be used as a basis for rejection of milk from unhealthy animals, a standard is needed. If this standard were as high as 1,000,000 leukocytes/ml., a considerable amount of milk would be rejected.

The appearance of milk is used as an indication of an abnormal condition of the udder, but by this procedure only a small proportion (about 8%) of mastitis is detected. Some milk producers use the strip cup for the detection of mastitis and believe that all animals having mastitis can be detected by their flaky milk. This is not true, since only about 26 per cent of animals with mastitis give flaky milk.

Mastitis test cards, or cards impregnated with bromthymol blue, are used by many milk producers for detecting mastitis. These cards will probably detect about 40 to 50 per cent of the cases; a positive test indicates a definite abnormality, but a negative test does not always mean that the animal does not have mastitis.

One objection to most of the tests for detection of mastitis is that they do not detect the condition in the early stages. The leukocyte count, perhaps, detects the condition much sooner than some of the other tests.

MASTITIS AND FARM SANITATION

The value of sanitation on the farm as a mastitis control procedure has been questioned by some, who believe that the organisms capable of causing mastitis are always present in the surroundings of a dairy farm and that it is not possible to eliminate them.

However, sanitation does seem to be important; the spread of mastitis from one animal to another has been reported and, in some cases, the infection has followed the sequence in which the animals were milked. Generally, good sanitary practices not only

help to prevent the spread of mastitis, but they are valuable in the production of milk with a low bacterial count.

As a precaution against the spread of mastitis, it should be assumed that the causative organism may be of a highly infectious nature and, therefore, it is desirable to: (1) eliminate or segregate animals with mastitis, (2) place them at the end of the milking line, and (3) disinfect the milking machine between animals.

Frequently, the latter is not properly done. The machine should be rinsed free of milk and treated with a bactericide after each animal is milked. Visual inspection will indicate if the rinsing was properly done. A common error is that insufficient contact time is allowed for the bactericide to destroy contaminating bacteria.

Improper administration of antibiotics has resulted in some cases of mastitis. One milk producer was employing a penicillin solution, which he prepared, that was heavily contaminated with yeasts. Penicillin bougies have been picked off the barn floor, when accidentally dropped, and inserted into the teat canal.

PROCESSING PROBLEMS

Milk from mastitic cows causes difficulties in milk processing. When homogenization of milk was first introduced, a sediment was noticed on the bottoms of some bottles. This sediment proved to be due to the leukocytes in milk. Consequently, it was necessary to replace less expensive milk filters with more expensive milk clarifiers to remedy this defect. A milk clarifier loses its efficiency when the bowl accumulates insoluble material such as leukocytes, precipitated casein, and dirt. Large processors alternate two clarifiers, so that one can be cleaned while the other is operating. Thus, milk processing costs are increased by mastitis.

Filtering milk on the farm is more difficult with mastitic milk. Today, with in-the-line filters, mastitis decreases the milk flow through such filters. On one farm, a clogged filter was responsible for an air leak at the filter and the air incorporation, plus agitation, resulted in a tank of rancid milk.

The resazurin test, used in some markets for assessing the bacteriological quality of milk, is influenced by leukocytes in the milk. As a result, milk with a high leukocyte count is degraded.

CONCLUSION

It is the opinion of many dairy bacteriologists, dairy fieldmen, and plant operators that milk quality can be increased considerably by the elimination of milk from cows with mastitis. The necessary legislation has been enacted to eliminate this milk. We need to know on what basis it is to be rejected, and then apply the test with sincerity.

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Leukopenia Associated with Acute Bovine Mastitis

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The purpose of this paper is to report the consistent observation of leukopenia in cows with experimental and field cases of acute mastitis.

The leukocyte counts herein reported are the lowest ones in a series made on each cow with experimental mastitis.

Counts reported on cows with field cases were made on single blood samples collected at the time each animal was observed. It was not possible to estimate the interval between time of infection and sampling in the field cases. For purposes of differential diagnosis, pretreatment milk samples were cultured from all affected cows.

CASE REPORTS

Acute Klebsiella (Capsulated Coliform) Mastitis.—Leukopenia was observed in all of 12 cows with experimental, acute Klebsiella mastitis. The average low count for the group was 3,025 per cubic millimeter, with a range of 1,350 to 4,200/cmm.

A typical leukocyte response is shown by an animal with experimentally induced acute Klebsiella mastitis (table 1). The

TABLE I—Typical Leukocyte and Differential Counts on a Heifer with Acute Experimental Klebsiella Mastitis

Time	Leukocytes*	Neutrophils*	Lymphocytes*	Monocytes*	Eosinophils*
Preinfection	7.81	2.20	5.30	0.23	0.08
2 hr.	8.88	1.80	5.50	0.35	1.23
4 hr.	4.78	1.50	2.90	0.19	0.19
6 hr.	2.16	0.28	1.70	0.09	0.09
9 hr.	2.00	0.18	1.78	0.02	0.02
12 hr.	2.00	0.14	1.80	0.06	0.00
24 hr.	2.20	0.22	1.85	0.04	0.09
2 da.	2.21	0.18	1.94	0.00	0.09
3 da.	3.61	0.28	3.19	0.00	0.14
4 da.	3.20	0.64	2.56	0.00	0.00
5 da.	4.90	1.03	3.72	0.00	0.15
6 da.	6.80	2.04	4.42	0.14	0.20
7 da.	6.90	2.07	4.83	0.00	0.00
10 da.	9.80	2.94	6.86	0.00	0.00
14 da.	10.71	3.75	6.96	0.00	0.00
21 da.	14.26	5.46	8.40	0.00	0.40

* = $\times 10^3$ cmm.

total count dropped from 7,810 to 2,000/cmm. within nine hours postinfection. The neutrophils dropped from 2,200 to 140/cmm. within 12 hours. The lymphocytes dropped from 5,300 to 1,700/cmm. within six hours. One cow with a field case of acute coliform mastitis had a leukocyte count of 5,800/cmm.

Acute Staphylococcal Mastitis.—In cases of experimental acute staphylococcal mastitis, leukopenia was observed in 3 of 3 cows. Their average low count was 3,716/cmm., with a range of 2,450 to 5,500/cmm.

In 14 cows with field cases of acute staphylococcal mastitis, leukocyte counts averaged 4,625/cmm., with a range of 1,300 to 9,300/cmm. Five cows had counts lower than 4,000/cmm. and 3 others (a total of 8 of the 14) had counts below 5,000/cmm.

Acute Pseudomonal Mastitis.—In 8 cows with field cases of acute pseudomonal mastitis, leukocyte counts averaged 5,170/cmm., with a range of 3,200/cmm. to 8,400/cmm. Three cows had counts lower than 4,000/cmm. and 2 others (a total of 5 of the 8) had counts below 5,000/cmm.

DISCUSSION

The higher leukocyte counts reported for cows with field cases of mastitis, as compared with experimental cases, may have resulted from the indefinite time of sampling in respect to the onset of acute mastitis.

The mechanism of leukopenia in acute mastitis is being investigated.

SUMMARY

Acute mastitis of coliform, staphylococcal, or pseudomonal origin, in cows, was accompanied by leukopenia early in the

course of the disease. There was also neutropenia, with relative lymphocytosis.

Nonmilking Due to Teat Injury

To prevent interference with healing of injured teats, from regular milking, the effect on the gland of retaining milk for several days was studied experimentally in normal cows. The unmilked quarter showed an increasing, painless tension which reached its climax in three days but, except in the later stages of lactation, this quarter milked normally at the end of the experiment. In the late stages of lactation, milking this quarter failed to restore normal lactation. Mastitis occurred in the unmilked quarter of only 1 experimental cow.

With rats, guinea pigs, and rabbits, when one gland was suckled regularly while all the others were covered with tape, normal involution of the nonsuckled gland was considerably delayed.

Apparently a ten-day interruption of milking to allow healing of teat wounds, whether sutured or not, does not disturb later lactation. However, before this is done, the injured gland should be examined physically and bacteriologically to ascertain that it was normal prior to injury. In all cases, interrupted milking should be accompanied by an intramammary infusion of an antibiotic preparation to aid in the control of bacterial development.—H. J. Heidrich in *Der prakt. Tierarzt* (April 1, 1958): 101.

When chicks were fed a manganese-deficient diet for six weeks, to produce perosis, the blood coagulation time was retarded to 8.7 minutes compared with 2.8 minutes for controls.—*Vet. Bull. (May, 1958): 1530.*

Bovine Mastitis Attributed to *Bacteroides* Species

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THERE WAS no history of acute mastitis in a herd of 43 Holstein-Friesian cows which were producing abnormal milk. Palpation revealed only a few udders with fibrosis. The dairyman's major complaint was that one filter disk was required to filter each cow's milk. The animals appeared in good condition.

BACTERIOLOGICAL FINDINGS

The results of a bacteriological examination of milk samples from these cows, done in January, 1956, by methods previously described,² were as follows: 56 quarters were infected with group B streptococci; 5 quarters with hemolytic, coagulase-positive micrococci; and 36 quarters with an unidentified encapsulated rod.

Thus, milk from 97 (57%) of the 172 quarters, involving 34 (79%) of the 43

laboratory using standard methods, were less than 30,000 per milliliter for three months prior to, and three months after, the time quarter samples were taken.

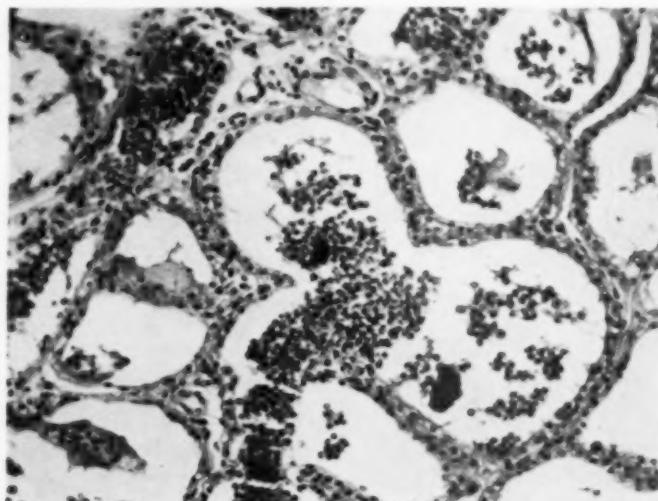
The encapsulated, rod-shaped organism isolated from 36 quarters was given particular attention. The milk obtained from these quarters was a viscous, clotted, stringy, curdlike secretion.

The colonies of this organism were small, moist, glistening, and mucoid on blood agar (blood agar base³) plus 5% citrated bovine blood.

Smears made directly from incubated Hotis samples revealed encapsulated organisms, whereas organisms of the colonies grown on blood agar were not encapsulated.

Growth in Brewer's semisolid thioglycolate agar was profuse in the depths, with none at the surface. Colonies in the semi-

Fig. 1—Acinar exudate, from a cow with *Sphaerophorus* sp. mastitis, consisting of polymorphonuclear leukocytes and calcific deposits. $\times 180$.



cows, contained pathogenic bacteria. The bacterial counts on samples of milk produced by cows in this herd, made in a

solid agar were soft and fuzzy but discrete, indicating nonmotility. After replating on Brewer's medium (1.5% agar) as a last check on purity, organisms isolated from 12 animals were studied in detail.

The cells were conspicuously pleomorphic, particularly those in carbohydrate medi-

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¹Produced by Difco Laboratories, Detroit, Mich.

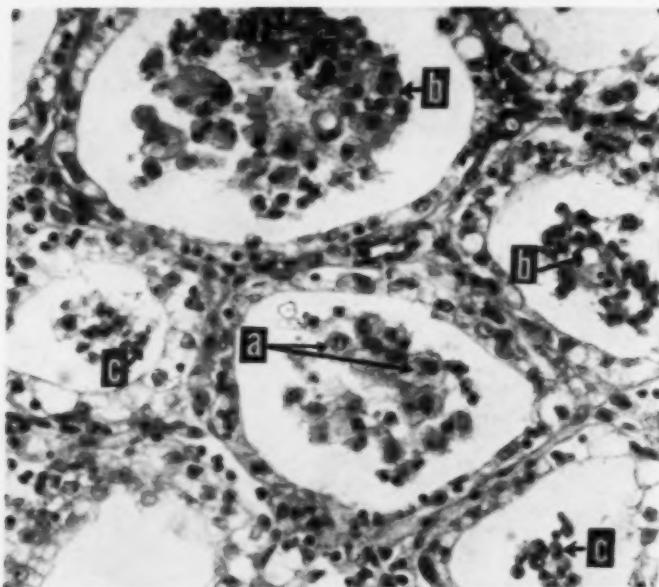


Fig. 2—Acinar exudate, from a cow with *Sphaerophorus* sp. mastitis, consisting of large mononuclear cells (a), an occasional sloughed epithelial cell (b), and polymorphonuclear leukocytes (c). Some of the epithelial cells lining the acini are vacuolated. $\times 350$.

ums. The pleomorphism was less pronounced among the encapsulated rods in the mastitic fluid. Cultural and biochemical tests were done by handling of the culture as an anaerobe. The organism proved to be a *Bacteroides* sp., probably *B. funduliformis* Hallé.³ (In the seventh edition of

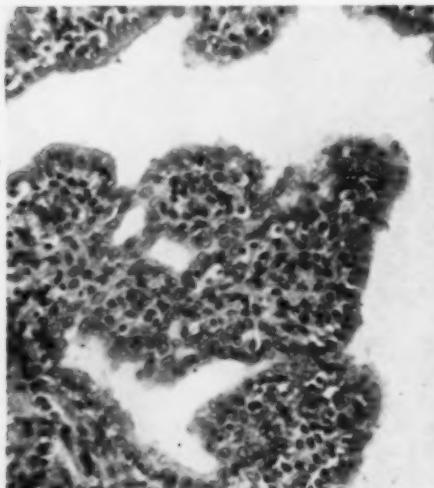


Fig. 3—Ductal epithelial proliferation, simulating intraductal papillomatous changes, in a cow with *Sphaerophorus* sp. mastitis. $\times 275$.

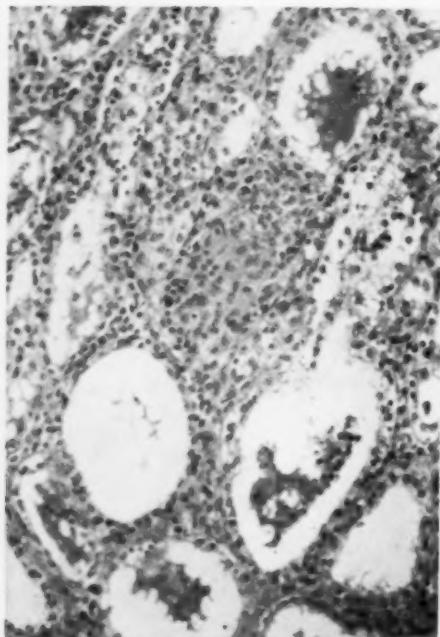


Fig. 4—Acinar displacement resulting from cellular infiltration and fibrous connective tissue proliferation, simulating early granuloma formation, in the mammary gland of a cow with *Sphaerophorus* sp. mastitis. $\times 180$.

Bergey's Manual, this name becomes a synonym of *Sphaerophorus necrophorus* (Flugge) Prévot.

The species identification must remain tentative for want of direct comparison with a known strain. The organism may be a saprophyte in the natural cavities of the animal body but can be a pathogen in otitis, pulmonary and liver abscesses, appendicitis, and genitourinary suppurations in man and abscess formation in rabbits and guinea pigs.³

Drug sensitivity studies were completed with the disk technique with 1 ml. of the thioglycolate broth culture as the inocu-

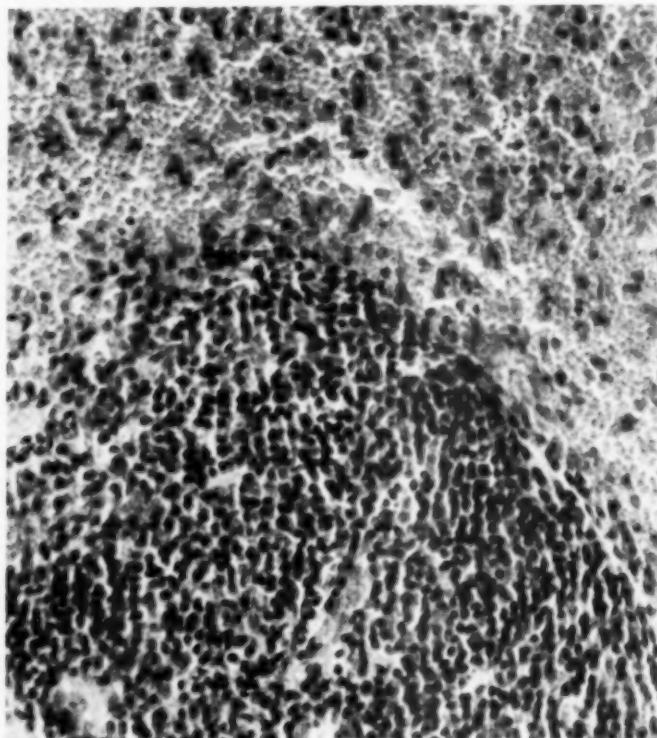
furazolidone (Furacin**), 100 µg. of streptomycin, and 10 units of penicillin. Unfortunately, the gram-negative rod was not encapsulated during the sensitivity studies. It would be difficult to extrapolate the disk sensitivity reactions to *in vivo* activity.

TREATMENT

Cows with infected quarters were treated in February and only the effect on the encapsulated rod will be reported.

The cows were allotted to two groups and treated as follows: group A—each infected quarter was infused with 10 ml. of oxytetracycline with polymyxin B sulfate; in group B—each infected quarter was in-

Fig. 5—Peripheral portion of a supramammary lymph node containing numerous polymorphonuclear leukocytes—from a cow with *Sphaerophorus* sp. mastitis.
x 700.



lum, with blood agar base as the medium in a poured plate. The respective disks were placed upon the agar surface. Sensitivity studies revealed that this organism was inhibited by disks of 10 µg. of chloramphenicol (Chloromycetin**), 30 µg. of chlortetracycline (Aureomycin**), 60 µg. of oxytetracycline (Terramycin**), 100 µg. of

fused as in group A and, in addition, was injected intramuscularly with 6 million units of crystalline procaine penicillin G in aqueous suspension.

**Chloromycetin is produced by Parke Davis & Co., Detroit, Mich.; Aureomycin by Lederle Laboratories Division, American Cyanamid Co., New York, N. Y.; Terramycin by Chas. Pfizer and Company, Inc., New York, N. Y.; Furacin by Eaton Laboratories, Norwich, N. Y.

The treatments were of limited value; 13 of 18 quarters of the cows in groups A and B were still shedding the organisms seven days following therapy, and failure to obtain growth from some quarters could be attributed to unfavorable growth conditions for the anaerobic organism and not necessarily to therapy.

Additional trials were conducted with the following drugs: streptomycin—5 Gm. per quarter, in a water-in-oil emulsion (HP Vehicle[†]) of neutral pH; nitrofurazone solution (Furacin Veterinary[†])—40 ml. per quarter;² SMP-1[†]—15 ml. per quarter; and intramammary tyrothricin suspension (Ty-sin[†])—40 ml. per quarter.

Therapy was essentially unsuccessful regardless of the material used. Many of the mastitic cows were marketed and the udder and a supramammary node of 1 was obtained for study.

PATHOLOGICAL FINDINGS

Gross Changes.—All four quarters of the mammary gland from this cow contained thick, cheesy milk. The glands contained small 1- to 2-cm. circumscribed lesions, believed to be either blocked ducts or small granulomata, which extended dorsally to the ligamentous attachment of the udder.

The node appeared normal in size, firm to palpation, and was resistant to cutting.

Microscopic Changes.—The acinar exudate was characterized by an infiltration of polymorphonuclear leukocytes (fig. 1) in some lobules while, in others, large mononuclear cells (fig. 2), which appeared to contain fatty material and debris, predominated. The acinus of an occasional lobule contained both acute and chronic cell types. In some lobules, the epithelium of the ducts was proliferated to the extent that it resembled intraductal papillomata (fig. 3). In many areas the ductal epithelium was vacuolated.

Lymphocytes, plasma cells, and an occasional eosinophil infiltrated the interstitial tissue. This and fibrous connective tissue proliferation caused acinar displacement and resulted in an occasional lobule resembling early granuloma formation (fig. 4).

The lymph node architecture was altered by a considerable proliferation of fibrous connective tissue, which resulted in the formation of islands of lymphocytes. An extensive, acute inflammatory in-

filtrate was also present. This change appeared more extensive toward the hilar portion of the node while, toward the periphery, aggregations of polymorphonuclear leukocytes (fig. 5) were interspersed with the lymphocytes.

DISCUSSION AND CONCLUSIONS

A peculiar mastitis affected 97 quarters of 43 cows (79% of the cows, 57% of the quarters), yet the bacterial count was only 30,000 per milliliter of milk. This paradox can be reconciled by a recognition of the fastidious growth requirements of the streptococci, and even more so of the *Sphaerophorus* sp. In a previous paper,¹ it was shown that there was no correlation between bacterial counts and the prevalence of mastitis.

The danger in attempting to translate data from determinations made *in vitro* to conditions *in vivo* is shown by the therapeutic failure of drugs which seemed effective on sensitivity tests. Their ineffectiveness may be explained on the basis of the capsulated versus noncapsulated states of the organism and inability of the drug to penetrate it in the affected udder. Studies have not revealed which metabolites are necessary, in artificial mediums, to cause the organism to remain encapsulated. Thus, the disk method of *in vitro* testing was not useful.

The gross and histopathological changes attributed to *Sphaerophorus* sp. mastitis are described.

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Bovine Mastitis in Finland

In ten years, reported cases of mastitis in Finland increased from 5,556 to 25,989 cases; about 15 per cent of 60,000 cows being affected. Most cases were due to *Streptococcus agalactiae*, but *Staphylococcus*, *Pseudomonas*, *Corynebacterium*, and *Actinobacillus* were also found. The control program consists of treating infected udders with antibiotics and disposing of carriers.—*Vet. Bull.* (May, 1958): Item 1386.

[†]HP Vehicle is produced by Hamilton Pharmacal Company, Inc., Hamilton, N. Y.; Furacin Veterinary is produced by Eaton Laboratories, Inc., Norwich, N. Y.; SMP-1 is essentially Chloramphenicol with a p-methylsulfonyl group substituted for the p-nitro group—code name product, produced by E. I. Du Pont de Nemours Co., Wilmington, Del.; Ty-sin is produced by Jensen-Salsbury Laboratories, Inc., Kansas City, Mo.

Polyvinylpyrrolidone* (HPX)—A New Treatment for Bovine Mastitis

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WE FIRST started cooperating with the New York State Mastitis Control Program in 1946 and, since that time, have continuously had from 40 to 50 herds of cattle under our supervision. We have accumulated thousands of clinical and bacteriological reports which represent a graphic study of mastitis treatments and results. As new antibiotics, sulfonamides, and steroid hormones have become available, we have given each a fair trial in our work. We should like to relate our experiences with a product, new for this purpose, which we have been using for some time.

Polyvinylpyrrolidone, an organic chemical derived from the high pressure synthesis of acetylene, was developed in Germany⁹ and was first used as a blood plasma expander in 1943.⁷ It was widely used by the Germans in treatment of civilian and combat casualties during World War II.

PHYSICAL AND OTHER PROPERTIES

Polyvinylpyrrolidone is a white amorphous powder, readily soluble in milk or water. Aqueous solutions are stable to heat and may be safely stored for long periods.¹² It is nontoxic whether given by oral administration, skin absorption, inhalation, or intravenous or intraperitoneal injection. The lethal dose is over 100 Gm. per kilogram of body weight. It is not a primary irritant, not skin fatiguing, and not a sensitizing material.¹³

In one investigation,¹⁴ 33 human patients were given 400 to 2,000 cc. of a 3.0 to 3.5 per cent solution as often as three times within 20 days without clinical manifestations of untoward effects. It was found¹⁵ to be nonantigenic and to cause no significant interference with liver or renal function after intravenous injection of quantities as large as 3,000 cc.

Polyvinylpyrrolidone seems to possess anti-inflammatory and detoxifying properties, the latter resembling those of natural blood plasma.¹⁶ It resembles plasma proteins both chemically and physiologically and has been shown to combine with, and render harmless, tetanus, botulinus, and diphtheria toxins.¹⁷ When 20 guinea pigs were injected with tetanus toxin, only 2 survived;¹⁸ how-

Drs. McAuliff, Phillips, and Steele are general practitioners in Cortland, N. Y.

*Polyvinylpyrrolidone is manufactured by General Aniline & Film Corp., New York, N. Y. Patents applied for by Hamilton Pharmacal Co., Inc., Hamilton, N. Y.

ever, when polyvinylpyrrolidone was given with the tetanus toxin, 12 of the 20 animals lived. It also reduced the mortality in mice when given following an injection of *Escherichia coli* endotoxin.¹⁹

It appears to reduce the toxicity and irritating effects of certain chemicals such as mercury, cyanides, and iodine. A solution of a complex which polyvinylpyrrolidone formed with iodine could be applied to the skin without any apparent irritation, even when bandaged,²⁰ and it reduced the toxicity of the iodine approximately tenfold.²¹ It was reported²² that polyvinylpyrrolidone often reduced or eliminated local inflammation following mammary infusion of large quantities of neomycin.

EXPERIMENTAL PROCEDURE

Polyvinylpyrrolidone seems to have two qualities that would be of value in mastitis treatments: ability to adsorb, or render harmless, certain potent toxins; and ability to reduce the irritating effects of such a highly toxic chemical as elemental iodine. We were supplied with a number of disposable syringes, each containing 1 Gm. of this product and, when infused into udders, it caused no irritation or other untoward effects.

Cows in four herds under the mastitis survey program, with infections as similar as possible, were used for bacteriological studies. The udders of those in two herds were infused with antibiotics only (group 1), and those in two similar herds were given antibiotics plus the polyvinylpyrrolidone (group 2). It was decided to limit the scope of this investigation to the most prevalent organism—*Streptococcus agalactiae*.

All quarters were checked by palpation and strip cup before samples were taken and any abnormalities were recorded. Milk samples were drawn by a veterinarian, using standard operating procedures, and were immediately taken to the laboratory for culture.

Mediums were prepared using sterile beef extract containing 2 per cent agar and 5 per cent bovine blood. Blood agar plates were prepared and incubated for 24 hours, at 37°C., immediately after preparation and prior to being used for the streaking of milk.

Milk was collected just prior to a regular milk-

ing, without discarding any before the sample was taken, and 0.01 cc. of this milk (no incubation) was streaked over one quarter of the blood agar plate. Milk from all four quarters of a cow was streaked on a single plate, which was incubated for approximately 24 hours at 37°C., then examined for evidence of bacterial growth. Streptococcus growths were transferred to a CAMP plate in order to distinguish *Str. agalactiae* from other streptococci. The CAMP plates were incubated for 24 hours at 37°C., then examined. *Streptococcus agalactiae* was identified on the basis of the presence of typical CAMP zones and the lack of esculin splitting.

TREATMENT

After receiving the laboratory reports, each infected quarter was treated. Infected quarters of the cows in group 1 were treated with 1 million units of penicillin and 1 Gm. of dihydrostreptomycin suspended in 20 cc. of emulsion; those in group 2 were treated with 1 million units of penicillin, 1 Gm. of dihydrostreptomycin, plus 1 Gm. of polyvinylpyrrolidone suspended in 20 cc. of emulsion. Lactating quarters, in the cows in all four herds, were treated twice in 48 hours; dry quarters were infused once.

RESULTS

In vitro studies have shown conclusively that penicillin at low levels will inhibit *Str. agalactiae*. However, the infusion of 1 million units of penicillin into 63 infected quarters in cows in group 1 did not eradicate

the infections whereas, in group 2, the incidence of *Str. agalactiae* was materially reduced (graph 1).

Also, in group 2, quarters that had previously been swollen and hard were definitely softening and inflammation seemed to be reduced to a marked degree. The herdsmen reported that these quarters "milked out easier than after previous treatments."

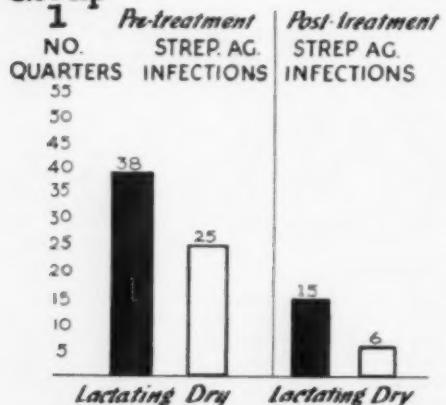
CONCLUSIONS AND SUMMARY

Polyvinylpyrrolidone (HPX), an organic chemical derived from the high pressure synthesis of acetylene, which had been used for many years as a blood plasma expander, was found to be nontoxic when infused into the bovine udder, even in large doses.

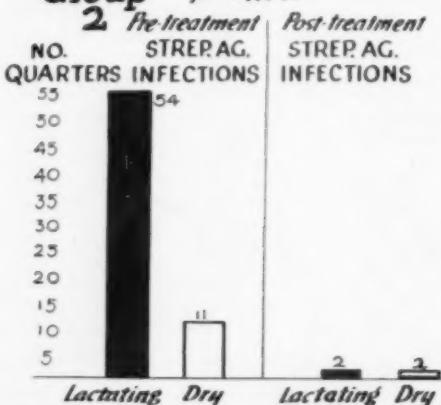
In cows in two herds, 63 quarters infected with *Streptococcus agalactiae* were infused with penicillin and dihydrostreptomycin with recovery, based on bacteriological evidence, in 67 per cent. In cows in two other herds, 65 similarly infected quarters were infused with a similar dose of penicillin and dihydrostreptomycin, plus 1 Gm. of polyvinylpyrrolidone, with recovery in 94 per cent.

Polyvinylpyrrolidone seems to possess a remarkable anti-inflammation action within the bovine udder. The reduction in swelling and inflammation apparently allows a more rapid and thorough penetration of antibiotics. (This product has also been reported to reduce the irritating effects of

Treated with antibiotics alone Group



Treated with antibiotics Group plus HPX



Graph 1—Results of treatment with antibiotics alone and antibiotics plus polyvinylpyrrolidone, in cows with *Streptococcus agalactiae* infection.

neomycin, so that large doses may be infused into a quarter, without untoward results.)

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Nocardia asteroides in the Mastitis Complex

Nocardia asteroides has been isolated from the mammary glands of 29 cows in California. Clinical manifestations of the infection have been observed in first lactation heifers within a week of calving, as well as in older animals. Infected glands produced altered milk which, in the more advanced cases, contained grossly visible white clumps of mycelia. The diseased glands were heavily fibrosed and often contained palpable nodules 2 to 5 cm. in diameter. The course of the disease was febrile, with temperatures reaching 107 F. While only a single fatality was observed, the extreme induration of untreated glands soon made slaughter an economic necessity.

Pathological changes in infected glands include large granulomatous lesions of the

parenchyma, in which the causative organism could be demonstrated, and proliferative changes of the ductile epithelium. The lining of the teat sinus was grossly thickened and granular. The lobular distribution of the pathological changes suggested an ascending infection via the teat canal. The disease was reproduced in an experimentally inoculated cow. Nocardia were isolated only from the milk and mammary tissue of the diseased animals.

Soil samples from the holding corrals were shown to harbor the organism.

The Nocardia in milk survived exposure to 64 C. for 30 minutes, but were killed by 66 C. in 30 minutes. Benzalkonium chloride and chlorine solutions at 200 p.p.m. were found to be the most germicidal of four disinfectant solutions tested.

A mixture of 500 mg. of novobiocin in 25 to 40 cc. of nitrofurazone (0.2%) solution was used successfully in cases of natural infection.

Tuberculin hypersensitivity resulting from *N. asteroides* mammary infections could not be demonstrated.—[A. C. Pier, D. M. Gray, and M. J. Fossatti: *Nocardia asteroides*—a Newly Recognized Pathogen of the Mastitis Complex. Am. J. Vet. Res., 19, (April, 1958): 319-331.]

Bovine Mastitis Due to *Pasteurella Multocida*

Spontaneous *Pasteurella* mastitis is rare in cows. Two cases are described. One was diagnosed by culturing the milk. Both hind-quarters were affected. Subcutaneous injections of cultures killed a mouse in two days and the organism was identified from heart blood.

In the second cow, one quarter had been affected for three weeks. At first, there were yellow mucous flakes in the milk which soon thickened until the secretion became lumpy and watery, with a bad odor. The quarter was swollen and sensitive but the cow's general condition was undisturbed. The milk showed an abundance of phagocytized *Pasteurella* as well as some *Corynebacterium pyogenes*, but the *Pasteurella* were considered the primary infection since the typical odor of *C. pyogenes* infection was not noticeable the first week.—H. Blum in Monatsch. f. Vet.-med. (April 15, 1958): 225.

Alimentary Toxemia in Chickens

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A NEW DISEASE appeared in chickens during 1957. Large numbers of birds from approximately 3 weeks of age to adult laying hens were affected. Clinical signs which have been observed by one of us (L.S.) in field outbreaks included dyspnea, reduced gains, stunting, subcutaneous edema, paleness, and sudden deaths. In young chickens, gasping was the first noticeable sign; this was followed by a waddling, unsteady gait in many. Clinical signs were much less noticeable in adults.

Feed consumption was never noticed to drop more than 10 per cent below normal in affected broiler flocks. In broilers, daily mortality averaged 1.0 to 1.5 per cent, with a total mortality of more than 50.0 per cent in some flocks. Adults were less severely affected and showed a wide variation in production and mortality rates.

The agent or agents which are causing

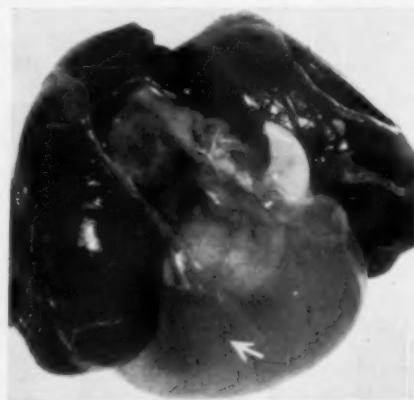
when certain samples of fat have been employed at levels of 2.0 to 7.5 per cent of the total ration.

The purpose of this paper is to present a study of the clinical signs and the gross and microscopic lesions in both early and advanced stages of the condition. Two groups of birds from different sources were available for examination. The chickens in one group showed early gross and microscopic lesions but no clinical signs; those in the second group were from a flock which had suffered from this disease for approximately eight weeks.

Gross lesions in birds in the early stages of this disease included pale hearts and an average of 0.6 ml. of fluid in each heart sac, and livers that were pale, mottled, and had an irregular granular surface with slightly depressed, dark intersecting lines over the surface.



Fig. 1—Broilers showing large distended abdomens filled with fluid, subcutaneous edema between the legs and body, and subcutaneous hemorrhages (arrows).



—Ohio Agric. Exper. Sta.
Fig. 2—Pericardium, from a chicken, distended with fluid (heart shadow at arrow) and with fibrinous membrane over part of the liver.

this disease have not been identified. No significant infectious agents have been recovered. Nutritional deficiencies and toxic substances have been suspected. In extensive laboratory tests, use of the common feed ingredients, medicaments, or stabilizers has not caused the disease. However, the disease has been readily reproduced

Broilers in the advanced stages of the disease had large, fluctuating, distended abdomens containing from 100 to 500 ml. of clear straw-colored fluid, large fibrin clots, and marked subcutaneous edema of the legs, abdomen, and breast (fig. 1).

The pericardial sac contained up to 20

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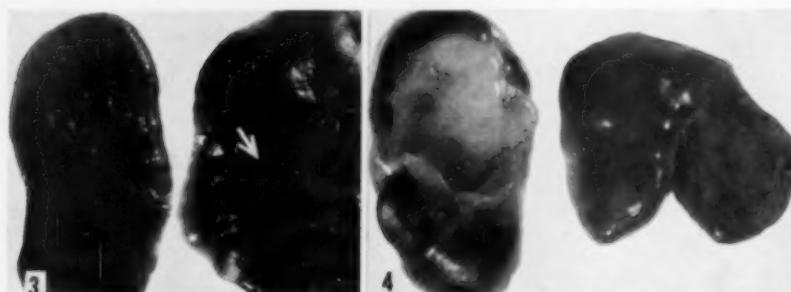


Fig. 3—Chicken livers, showing irregular grayish areas of necrosis (arrow) and granular appearance of the surface.

Fig. 4—Chicken livers, showing swollen irregular lobes with fibrinous membrane covering the surface.

ml. of clear, straw-colored fluid (fig. 2). The heart was pale and, in some birds, the epicardium had a whitish appearance. The livers were swollen and had rounded edges, mottled light and dark colored areas, and irregular roughened surfaces caused by a slight depression of intersecting lines over the entire surface (fig. 3); others were nodular, firm, and friable, with a nutmeg or bronze color. Most of the livers had a removable grayish, fibrinous membrane adhered to the surface (fig. 4).

Eccymotic hemorrhages were present in the skin beneath the wings, on the legs, and over the keel bone in a few chickens (fig. 1). Some hearts had petechiae and ecchymoses in the myocardium, on the auricles, and under the epicardium, but gross hemorrhages were not a prominent feature of this disease.

The kidneys were pale and swollen. Gizzards showed edema of the fatty tissue and

the duodenum appeared swollen, soft, and edematous.

LABORATORY PROCEDURES

Abdominal and pericardial fluids were collected aseptically and cultured in nutrient broth and on blood agar, but no bacterial growth developed. Cultures of liver, heart, and spleen did not yield any significant organisms. A few contaminating *Escherichia coli* and micrococci were recovered. Tissues and fluids were checked for the presence of PPLO and psittacosis agents but none were found.

Intestinal washings, diluted with sterile physiological saline solution, and pericardial and abdominal fluid were injected intravenously into white mice but the mice survived with no evidence of toxicosis. Entire intestinal tracts were homogenized in a Waring blender, diluted with sterile water, and fed to white mice and 4-week-old

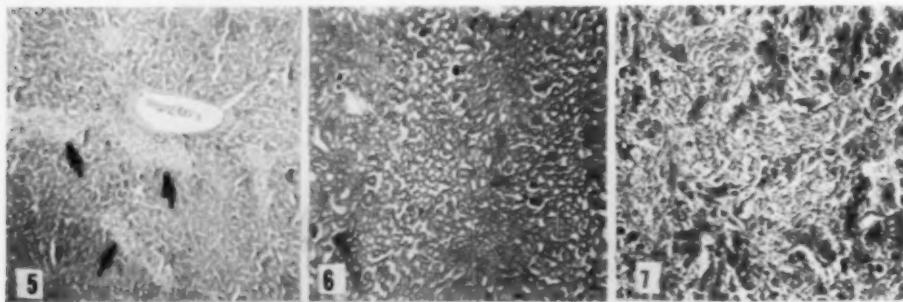


Fig. 5—Early lesions of focal necrosis (arrows) around the central vein in the liver of a chicken. H & E stain; x 24.

Fig. 6—Early lesions in the kidneys of a chicken, showing interstitial edema and distended Bowman's spaces. H & E stain; x 24.

Fig. 7—Extensive hepatic fibrosis in a broiler which had been affected with alimentary toxemia for several weeks. Trichrome stain; x 38.

broilers for several days, but no ill effects were observed.

HISTOPATHOLOGY

Hematoxylin-eosin stained sections of liver, heart, spleen, lung, kidney, duodenum, pancreas, testicle, bone marrow, gizzard, sciatic nerve, brain, skeletal muscle, and adrenal gland were studied.

Early lesions were limited to the liver, kidney, adrenal gland, and cerebellum. There were small foci of necrosis and degenerating cells in the liver (fig. 5). Pectechiae were numerous and areas of poorly stained cells were seen, indicating early de-

generative changes. Liver lobules were intact and there was no increase in fibrous tissue.

Epithelial cells in the proximal convoluted tubules of the kidneys showed albuminous degeneration. Henle's loops were unaffected, glomerular tufts were swollen, and detail was indistinct and Bowman's spaces were dilated. Erythrocytes were lying free in some tubules and in Bowman's spaces. Tubules were separated by open spaces, indicating interstitial edema (fig. 6).

In the adrenal gland, petechiae and ecchymoses, as well as edema, were present in the interstitial spaces and albuminous degeneration was apparent

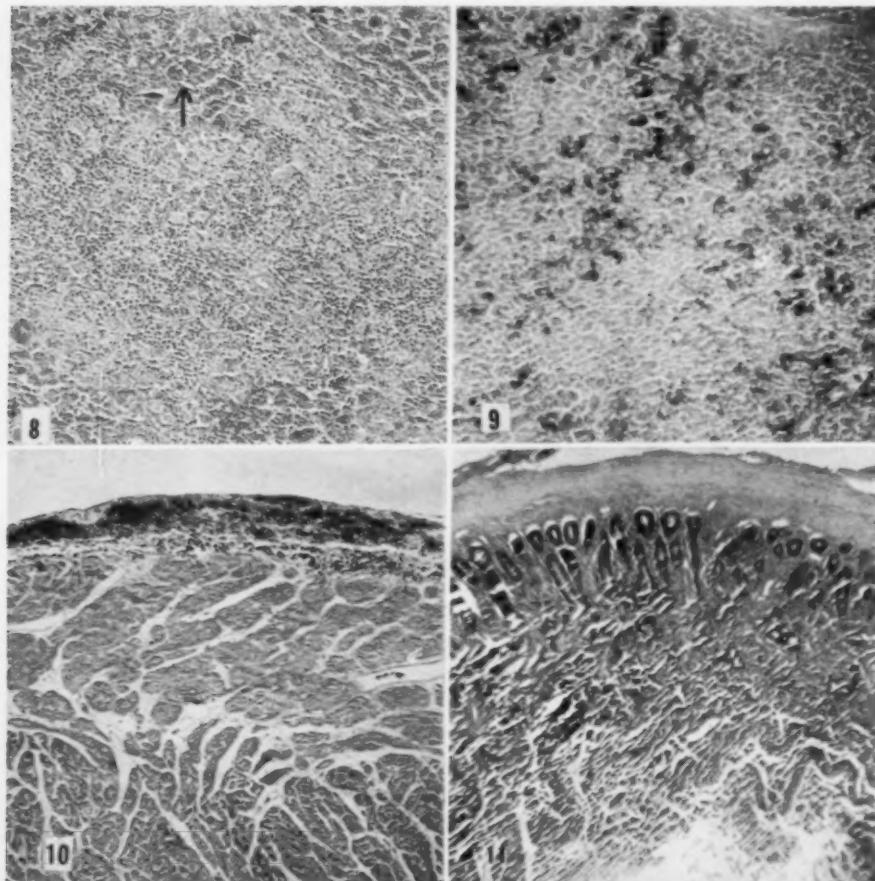


Fig. 8—Tissue from broiler that had been affected with alimentary toxemia for several weeks showing extensive liver necrosis with infiltration of heterophils. Groups of degenerating liver cells remain (arrow). H & E stain; x 80.

Fig. 9—Chicken liver showing extensive hemorrhages. H & E stain; x 43.

Fig. 10—Subepicardial hemorrhage and edema in affected chicken. H & E stain; x 80.

Fig. 11—Small intestine of chicken showing necrosis of the peripheral layer of the mucosa. H & E stain; x 24.

in some parenchymatous cells. Purkinje cells in the cerebellum were undergoing degeneration. Some were swollen, pale, and vesicular; others remained only as small eosinophilic masses.

In the more advanced condition, lesions differed mainly in degree from the early lesions. In the most severely affected chickens, nearly all liver cells were destroyed, necrosis of entire lobules had occurred, many heterophils were present (fig. 8), petechiae were numerous throughout the tissue, fibrosis (fig. 7) was prominent, and liver architecture was partially destroyed. Many sinusoids were destroyed. Small bile ducts could be seen in these areas, with the epithelium undergoing albuminous degeneration. There was no bile duct hyperplasia and no evidence of regeneration of liver cells.

In livers from chickens less severely affected, lesions were correspondingly less extensive, although petechiae were numerous (fig. 9).

In the heart, focal subepicardial hemorrhages and lymphocytic infiltrations were prominent. The epicardium was separated from the myocardium by the hemorrhage and edema (fig. 10). Myocardial fibers showed some degeneration.

Echymoses were present in the interstitial tissues near the center of the adrenal gland.

The tips of the villi in the duodenum showed necrosis (fig. 11) but the deeper layer of the mucous membrane and the wall were unchanged. In 1 bird, there was necrosis of all pancreatic cells at the free edge of the gland. The cells beneath the area of necrosis were detached, rounded, and shrunken. Beneath this layer, the tissue was unaffected.

Hyaline casts and fibrin were present in some kidney tubules—others were widely dilated. A few glomerular tufts were detached, shrunken, and lying free in large, clear Bowman's spaces but Bowman's capsules were unaffected. No brain tissue was available from these broilers and other tissues showed no significant changes.

DISCUSSION

According to all reports, this problem appears to be confined to chickens, and its effect is not limited to one age group. No description of such a condition in chickens was found in the literature, although numerous reports described some aspects of vitamin E deficiency and salt poisoning which resembled this disease and which could be reproduced under experimental conditions.

Salt poisoning in chickens and poult has been studied by many workers but none reported^{1,3,5,12,13,17,18,20,21,24,25} the extreme liver necrosis in salt poisoning that was found in this disease. Also rather high levels of salt (3% or more) must be fed to cause toxicosis. Turkey poult are equally

susceptible to salt poisoning and high sodium concentrations but the authors have not seen this condition in turkeys.

Vitamin E deficiency has also been reported by many authors.^{6,7,8,10-12,15,16,19,23,26} Some lesions of vitamin E deficiency and this new disease are similar, but the extensive liver changes that were found here have not been described for vitamin E deficiency. Encephalomalacia was not found, nor has it been reported from the field; neither was muscular dystrophy observed in these chickens.

In field studies by one of us (L.S.), 10 units of vitamin E added per pound of feed did not prevent this condition. In rats on vitamin E- and cystine-deficient diets, liver necrosis had been prevented by feeding either vitamin E or cystine.²²

Highly unsaturated fatty acids contributed to signs of increased capillary permeability in 1-day-old chickens on a vitamin E-deficient diet, but rancidity of the dietary fats did not contribute to development of this disease.⁹

The effects of numerous chemical and plant poisons have been studied in chickens.^{2,6,16,27,28} In some instances, lesions have been described which resembled some of the changes seen in this condition but no description was found that closely paralleled the lesions reported here.

A careful study of the histopathological changes will probably be necessary to differentiate this condition from other entities which appear to be somewhat similar.

Only a limited number of fat samples have been encountered which were capable of producing the disease. It may be significant that none of the fat samples tested, which were obtained directly from primary rendering or extraction processes, produced the condition. It may be that the fats which caused the disease were altered in some manner.

Considering the small percentage of fat added to poultry rations and the limited quantity of feed consumed by a chicken in the first three weeks, it appears that the causative agent must be extremely toxic to produce the extensive lesions observed in this condition.

Feed known to be carrying the toxic agent has been mixed with equal parts of ground corn and fed to fattening hogs for 60 days, with no apparent harmful effects; the animals made excellent gains.

SUMMARY

A new disease appeared in broiler and laying chickens in 1957. Clinical signs in broilers included dyspnea, wobbly and unsteady gait, stunting, subcutaneous edema, and sudden deaths. Over 50 per cent mortality occurred in many flocks.

Gross lesions included ascites, subcutaneous edema, hydropericardium, hemorrhages, fibrinous membrane on the liver, and swollen, pale, irregular livers. Microscopic lesions included subepicardial hemorrhage and edema, degeneration of the myocardium, extensive hepatic necrosis with fibrosis, petechiae in the liver, hemorrhages and edema in the adrenal gland, and degeneration in the kidney in the more advanced stages of the disease. Clinical signs, egg production, and necropsy lesions were quite variable in adult birds which were exposed to the toxic substance.

This disease somewhat resembles acute salt poisoning, vitamin E deficiency, and poisoning by some chemicals and plants. However, no description was found in the literature which completely paralleled the condition described here.

The common ingredients of poultry rations, medicaments, and other additives have not produced the disease in laboratory tests, but certain samples of fat or feed containing this fat have caused the condition.

Since no sample of fat obtained directly from the primary rendering or extraction process has caused the disease, it may be that the troublesome fats have been changed in some manner from their original nature.

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Are We Using the Right Approach to Control Mastitis?

GUEST EDITORIAL

FEW DAIRYMEN will deny that the most costly disease which they observe in their cows is mastitis. However, there is one other disease which, I believe, costs them more in the end but which is certainly much less obvious. I refer to what is facetiously termed "hollow gut"—essentially, the inability of the ruminant digestive scheme of animals to obtain sufficient nutrients from inferior grade roughages to maintain the superior production for which present day dairy animals are selected.

We all know that animals will not consume enough additional poor grade roughage to make up for its deficiency in quality. Nor can we expect a digestive system developed for handling roughage to completely adapt itself to the substitution of increasing quantities of the good concentrates to which our feed industry is geared.

Both of these adaptation problems may have become accentuated with the intensified selection of higher producing animals and their concomitant necessity for greater food utilization to maintain their higher milk yield. Greater production and lower production costs have not been obtained for nothing, and part of the price has been an increase in the prevalence of certain types of diseases, one of which is mastitis.

NEED FOR RESEARCH

It has usually been necessary to determine the cause of a disease before adequate measures for its eventual elimination can be developed. Herein lies the difficulty with bovine mastitis. There are sincere differences of opinion as to what constitutes its cause, or causes, and what relative importance should be assigned to those non-specific circumstances which seem to predispose the malady. These differences of opinion are the reasons why more research is essential if we are to solve this riddle.

That there is confusion regarding the cause or causes of this disease is indicated by the recent survey of practitioners in Pennsylvania.² Of the 528 veterinarians sent questionnaires in Pennsylvania, 148 (28%) replied. Of these, 4 per cent agreed that "mastitis is an infectious disease

caused by organisms not found in normal animals, with effective control demanding isolation and treatment or elimination of carrier animals"; 20 per cent believed that mastitis "results when certain stresses overcome resistance to udder infection with certain management practices having greater influence on the control of the disease than any laboratory control program"; and 69 per cent were of the opinion that neither definition was adequate, and that "all or parts of both explanations must be included." I agree with the 69 per cent. We need similar surveys in other areas.

To the confusion of defining the cause or causes of mastitis must be added these circumstances:

1) There has been considerable good research on infectious agents as causes of mastitis, but there has been a paucity of research in the area of defining, by actual measurements, those nonspecific circumstances which produce changes in the cow herself, or in her udder, which may at least predispose the microbial invasion when it occurs. This one-sided research for 25 years has done little more than continue the inadequacy of our present knowledge as to why cows have mastitis.

2) There are a multitude of infectious agents reported to be associated with mastitis, some of considerably greater prevalence than others. This suggests the possibility that an infectious agent is not necessarily the primary cause of mastitis and that other factors may precede infection.

Pasteur believed that the physiological state of the infected individual often decided the course of an infection. He also suspected that such factors as the mental, genetic, and nutritional states played a role in resistance to disease. These factors have been given too little attention in many experimental studies in recent years.

3) There is a paucity of information on reproducing mastitis with various organisms isolated from mastitic udders. The difficulty in satisfying Koch's postulates also should suggest that something must precede infection when it is present.

4) There seems to exist a large percentage of mastitic cows from which organisms have not been isolated. Of 15,455 affected quarters examined in Scotland during the

This editorial is based on a paper presented by Dr. Nichols at the symposium on mastitis, Edgewater Beach Hotel, Chicago, Ill., February, 1958.

years 1951 to 1954, 54.3 per cent were non-specific, *i.e.*, "without demonstrable bacteria."⁵ What kinds of specific antibiotic therapy should be used on these? Or what tests should be used for their detection?

5) There seems to be an ever-increasing feeling that, among the many so-called "nonspecific" stresses, machine milking has contributed to greater difficulty with this disease, not alone as a tool for spreading organisms, but also one which increases functional damage of the udder and its teats. There is, however, a complete lack of data on the production of measurable stress by different types of machines—also on the practices used with machine milking. The milking machines and practices should be designed to more nearly fit the requirements of the cow, rather than to try to adapt the cow to fit the mechanical limitations of machines or the frailties of human nature so often observed in their use.

Here is an area where intense coordinated efforts among physiologists, pathologists, and engineers could be most fruitful. This problem is not a simple one, nor is it an unsolvable one. If we have the imagination and brain power to orbit satellites, we should be able to solve this problem in mechanics. The criterion of a successful machine is more than ascertaining that the teat cups fit; it must also be determined that it does not cause stress beyond the cow's ability to adapt to it.

6) We now know that this disease does constitute a measurable stress. What was considered an increase in plasma levels of the adrenal hormone, hydrocortisone, during mastitis has been demonstrated.¹ A section of our laboratory has substantiated these hypotheses and techniques. One of our aims of the future is to attempt to differentiate predisposing stresses from those which occur during the courses of several diseases, including mastitis. Success will depend on the financial support mustered. Such research is complex and expensive.

We need the measurable data to define those practices which actually cause stress, not those that we merely think or guess are stress factors. We need the same techniques as measurable yardsticks to demonstrate whether those practices which we recommend for control actually do control predisposing stresses. To my knowledge, no one presently has this kind of data, and most of those factors which we loosely call "predisposing stresses" are unproved.

DISCUSSION AND CONCLUSION

What is our philosophy toward this disease going to be? It seems that one of the newer philosophies of mastitis control, practiced particularly by those who do not own dairy cows, is silence. Dairymen will not accept such a solution.

Another short-sighted philosophy is for one group to try to gain favor for its singular point of view without including all possible facets or phases of the problem which other groups may feel are equally important. One might compare this philosophy with adding more to the steeple while the termites eat away the foundation.

There is no question that improved methods of treatment are doing a good job and that even better treatments need to be developed—but are we to throw up our hands and say that we are stuck with mastitis—that the only thing we can do is devise new ways of handling it after the cows become ill?

Our extension people have accepted this challenge and are trying to teach dairymen to use what preventive information we have. It is now up to our research people to accept the challenge also, and to answer the following questions:

- 1) What are the measurable stress factors, both specific and nonspecific, which result in mastitis?
- 2) How can we control them?
- 3) Can we breed cows more capable of withstanding these stresses?
- 4) How far will our present information go toward eliminating this disease?

Until these questions are answered by concerted research, it is questionable whether this disease can be eliminated. I believe that, in the end, our cheapest way out of this dilemma is research by people trained for research, and with an approach to this aspect much broader than it is now. I believe we have no moral right to admit defeat with the disease and saddle our future with techniques for its continuation.
—R. E. Nichols, D.V.M., University of Wisconsin, Madison.

References

- 'Bailey, W. W.: Prednisone (Meticorten) as Supportive Therapy in Stress Conditions in Large Animals. *J.A.V.M.A.*, 132, (Jan. 1, 1958): 27-30.
- ⁵Buss, S. B.: Survey of Pennsylvania Veterinarians on Mastitis and the Antibiotics Problem. *Vet. Med.*, 53, (Jan., 1958): 50-52.
- ¹Laing, C. M., and Malcolm, J. F.: The Incidence of Bovine Mastitis with Special Reference to the Non-Specific Condition. *Vet. Rec.*, 68, (1956): 447.

Current Literature

ABSTRACTS

Renal Studies on Cattle

A limited study of renal function in normal dairy animals and in cows with atypical paresis was reported. In the paretic animals, the effective renal plasma flow and "perfusion fraction" tended to be reduced and the "filtration fraction" increased, as compared with the normal postparturient animals. The paretic animals had low mean blood pressures, low pulse pressures and, in some cases, atrial fibrillation and cardiac dilatation.

Of the examinations and analyses performed, no biochemical changes or changes in the blood cells were found in the paretic animals, as compared with the normal postparturient cows. The adrenal gland responses to injected adrenocorticotrophin and the appearance of the adrenal glands, cytologically, were similar in the control and paretic animals.—[A. F. Sellers, W. R. Pritchard, A. F. Weber, and J. H. Sautter: *Renal Function Studies on Normal Dairy Cattle and Those with Postparturient Albuminuria*. *Am. J. Vet. Res.*, 19, (July, 1958): 580-584.]

Spirilla in Dogs and Cats

Spirilla (*Spirillum rappini*, *Spirilla canis*) were found in the fundic regions of the stomachs of normal adult dogs (15) and cats (12), but not in 3 kittens. The microorganisms were located principally in the glandular lumina and in the intracellular canaliculi of parietal cells, showing an exclusive predilection for the latter cell. The spirilla appeared to be pathogenic rather than saprophytic, in that these forms seemed to be involved in a progressive enlargement of intracellular canaliculi with eventual degenerative changes in the cytoplasm and nuclei.

Living spirilla were isolated from fundic mucosal scrapings and observed under dark-field illumination. Sizes and shapes of spirilla and some of their structural characteristics and staining properties also were discussed.—[Alvin F. Weber, Osman Hasa, and Jay H. Sautter: *Some Observations Concerning the Presence of Spirilla in the Fundic Glands of Dogs and Cats*. *Am. J. Vet. Res.*, 19, (July, 1958): 677-680.]

Therapy Against *Dirofilaria immitis*

Antimony, in twice the microfilaricidal dose, was shown to prevent the development of *Dirofilaria immitis* in experimentally infected dogs. This was the first demonstration of prophylactic action by any chemical. It was suggested that both diethylcarbamazine and the arsenicals be similarly evaluated. Particular stress was placed on the arsenicals because they are more active against the adult worms than either diethylcarbamazine or antimony. It was pointed out that developing worms biologically are more nearly like adult worms than microfilariae.—[Seiji Kume: *Prophylactic Therapy Against the Developing Stages of*

Dirofilaria immitis Before Reaching the Canine Heart. *Am. J. Vet. Res.*, 19, (July, 1958): 675-676.]

Effects of Radiation on Hog Cholera Virus

Hog cholera virus (HCV) in swine blood serum preparations was irradiated with ultraviolet (2,537 Å) radiation. Approximately 10^5 minimum lethal doses (m.l.d.) of HCV, when ultraviolet-inactivated, was found to protect swine, 2 to 4 months old, to a 5,000 m.l.d. challenge. Reactivation of the infectivity of two of three HCV preparations was found to occur when ultraviolet doses appreciably in excess of the initially inactivating dose were used.—[Kenneth E. Collins: *Effects of Ultraviolet Radiation on Hog Cholera Virus*. *Am. J. Vet. Res.*, 19, (July, 1958): 540-544.]

Enteroviruses of Swine

A total of 376 agents cytopathogenic in porcine kidney tissue culture have been isolated from the feces of a population of healthy pigs over a one-year period of study. Of 123 strains to date, 121 have been shown to be antigenically related to a prototype, indicating that the majority belong to a major homotypic group.

The agents, characterized as viruses, multiply readily only in tissue cultures of porcine origin and are innocuous to common laboratory animals.

Preliminary studies in swine indicate that the virus actively infects pigs during the early months of life and that infected pigs continue to shed virus in the stools. This study adds to the existing evidence that viruses are present in domestic animals without associated clinical illness.—[George W. Beran, Alvar A. Werder, and Herbert A. Werner: *Enteroviruses of Swine. I. Their Recognition, Identification, and Distribution in a Herd of Swine*. *Am. J. Vet. Res.*, 19, (July, 1958): 545-553.]

Growth Response of *Salmonella Pullorum*

As little as one organism was found to constitute a minimum lethal dose of *Salmonella pullorum* for embryonating eggs upon intra-allantoic inoculation.

It was further established that the growth rate for *S. pullorum* in the allantoic fluid was exponential, the maximum approaching 10^9 organisms per gram of allantoic fluid. Some fluctuation in the number of bacteria in the allantoic fluid was encountered at 12 to 18 hours postinoculation. At the same time, the bacterial population in the embryo increased at a rapid rate.

Two media were used for growth of *S. pullorum*. Little difference was observed in the pathogenicity of bacterial cells from the synthetic and complete media.

These observations indicate the importance of using clean eggs in hatcheries.—[C. D. Jeffries and D. F. Holtzman: *Growth Response of Salmonella Pullorum in the Chicken Embryo*. *Am. J. Vet. Res.*, 19, (July, 1958): 736-739.]

THE NEWS

Veterinary Congress to Be Held in Brussels, September 6-7

An announcement has been received of a Veterinary Congress scheduled to be held in Brussels, Belgium, Sept. 6-7, 1958, as one of the events taking place during the Brussels' World Fair and International Exposition currently in progress.

This congress is under the joint patronage of the Belgian ministries of agriculture, public health and welfare, public instruction, and colonies; its program is also sponsored by the L'Union Vétérinaire Belge (Belgian Veterinary Association).

Distinguished authorities in veterinary science will appear on the two-day program to discuss the role of veterinary medicine and science in social hygiene and public health and in improving agriculture. They will also examine the role of animal production in the economic and social evolution of man and the role of the state in the economic production of animals.

Persons interested in attending this congress may obtain information about registration fees, social functions, and hotel accommodations by writing to: Bureau—Journées Vétérinaire Bruxelles 58, 35 Rue de la Brasserie, Bruxelles 5, Belgium.

The Fourth International Congress on Animal Reproduction Postponed Until 1961

The organizing committee in the Netherlands for the Fourth International Congress on Animal Reproduction has, in cooperation with the members of the Executive Committee, decided to postpone the next congress from 1960 to 1961.

The first consideration in changing the initial meeting date was the fact that the next Congress on Animal Production will be held in 1961, not in 1960, and the International Committee decided in Cambridge in 1956 that there should be coordination between the Congress on Animal Reproduction and the other Congress.

Secondly, the Congress on Fertility and Sterility, which will meet in Amsterdam in 1959, has a rather large veterinary section. (This congress is sponsored by the International Fertility Association and deals mostly with human fertility and sterility.)

This meeting should be of interest to veterinarians, pathologists, and physiologists who are engaged in research on reproduction.

Second Poultry Disease Diagnostic Short Course for Regulatory Veterinarians Held at Iowa State

The second poultry disease diagnostic short course for regulatory veterinarians was held at Iowa State College by the Division of Veterinary Medicine April 14-25, 1958. This is the first time that state and federal veterinary regulatory agencies have joined forces with a



Regulatory veterinarians who attended the second poultry disease diagnostic short course at Iowa State College stand at the entrance of the new veterinary diagnostic laboratory where most of the work was presented.

Front row (left to right)—Paul Nicoletti (Mo.); M. J. Eggert (Va.); P. H. Seitz (Pa.); Gabriel Gonzalez (P.R.); C. D. Lee (Ames); Dean I. A. Merchant, (Ames); R. E. Omohundro, (Washington, D.C.); C. L. Vickers, (S. Car.); L. D. Lewy, Jr., (La.); J. D. Winward, (Utah); A. M. Jasmin, (Mont.); John Hudelson, (Kan.); A. E. Crawford, (Ind.); H. L. Arnold, (Okla.)

Back row (left to right)—Drs. W. E. Clarke, (Ga.); C. A. McDonald, (W. Va.); A. A. Heflin, (Mex.); L. E. Bodenweiser, (Ore.); P. D. Fichandler, (Conn.); John F. Long, (Ohio); Leroy Pierce, (Iowa); H. P. Hickox, (Mo.); J. J. Woolsey, (Ariz.); C. V. Simmons, Jr., (Ill.); J. G. Wadsworth, (Ark.)

veterinary school to give "in service" specialized training. Similar courses are planned for the future.

Ten veterinarians employed by ten different states attended the course, along with 11 veterinarians from the Animal Disease Eradication Division of the Agricultural Research Service, U.S.D.A., and one veterinary circuit supervisor from the Inspection Branch of the Poultry Division of the Agricultural Marketing Service.

The arrangements for the short course were made through Dr. C. D. Lee, extension veterinarian, and Dr. I. A. Merchant, dean, Division of Veterinary Medicine, Iowa State College, at Ames.

The purpose of this training is to make specialists available for differential diagnostic work in cooperation with reference laboratories established for coping with an outbreak of foreign poultry diseases or unusual domestic outbreaks. Veterinarians from their respective states will act as poultry disease diagnosticians.

AMONG THE STATES AND PROVINCES

Arkansas

State Association.—The following officers are serving the Arkansas V.M.A. for the ensuing year: Drs. John C. Smith, Stuttgart, president; James L. Forgasen, Morrilton, vice-president; and H. M. Goins, Berryville, secretary-treasurer. *s/H. M. GOINS, Secretary-Treasurer.*

California

Santa Clara Valley Association.—The Santa Clara Valley Veterinary Association recently elected the following officers for the ensuing year: Dr. Loris Johnson, San Jose, president; Robert Olsen, San Jose, vice-president; and Robert King, Santa Clara, secretary-treasurer. *s/R. L. KING, Secretary-Treasurer.*

Colorado

Dr. Herin Awarded National Science Foundation Postdoctoral Fellowship.—Dr. Reginald A. Herin (COL '58) has been awarded a National Science Foundation Postdoctoral Fellowship to do research work in physiology. Dr. Herin will work on "The Thermo-Regulatory Effects of the Abdominal Air Sacs on Spermatogenesis in Domestic Fowl."

His research will consist of determining the cooling effects of the abdominal air sacs on the testes. Dr. Herin will work under the direction of Dr. Nicholas H. Booth (MSC '47), head, Department of Physiology, College of Veterinary Medicine, Colorado State University, Fort Collins.

Prior to attending Colorado State University, Dr. Herin attended Millsaps College and the U.S.A.F. Air Institute of Technology. He



Dr. Reginald A. Herin

served 11 years with the U. S. Air Force as a jet pilot and contracting officer.

s/N. H. BOOTH, Head, Department of Physiology, Colorado State University.

Indiana

Women's Auxiliary.—The Women's Auxiliary to the Michiana V.M.A. held its biennial meeting in the Empire Room of the Hotel Elkhart in Elkhart, on May 14, 1958, during the biennial clinic of the Michiana V.M.A.

A representative of the Lenox China Company, Trenton, N. J., discussed fine china and showed a movie on its manufacture. Among



Mrs. A. L. Starkey (left), Mrs. W. W. Armistead, Mrs. J. J. Fishler, Mrs. L. M. Hutchings, and Mrs. M. J. Klooster.

the auxiliary's special guests were: Mrs. W. W. Armistead; Mrs. L. M. Hutchings; Mrs. M. J. Klooster, president, Auxiliary to the Michigan V.M.A.; and Mrs. A. L. Starkey, president, Auxiliary to the Illinois V.M.A.

The committee in charge of making the arrangements for this meeting were: Mrs. J. J. Fishler, chairman; Mrs. F. R. Booth, Mrs. J. M. Carter, Mrs. A. C. Dufour, Mrs. W. G. Magrane, and Mrs. C. M. Poole.

Iowa

Dr. Monlux Is Honored by His Students at ISC.—The Iowa State College Student Chapter of the AVMA presented an award to Dr. William S. Monlux (ISC '37), Division of Veterinary Medicine, for his outstanding work in their behalf at the chapter's annual awards meeting, June 4, 1958.

The award, which was made possible by the voluntary contributions of Dr. Monlux's various classes, took the form of a 12-cubic-foot deep freeze.

In presenting the award, the students noted that their professor spends hours preparing tape recordings of his lectures to make them both informative and interesting. They further



Dr. William S. Monlux stands beside the deep freeze which his students at Iowa State College presented to him June 4.

stated that they consider his teaching and personality invaluable motivating factors in their practice of veterinary medicine.

Dr. Monlux is the author of a number of papers dealing with veterinary medicine. He is a member of Alpha Psi, Phi Kappa Phi, Phi Zeta, and Sigma Chi. He joined the faculty at Iowa State in March, 1953.

S/BILL SPEER, President.

North Carolina

Dr. Hines Receives Reynolds Public Health Award.—Dr. Martin P. Hines (OSU '46), North Carolina State Board of Health, received the Carl V. Reynolds Award on May 30, 1958, at the meeting of the North Carolina Public Health Association, the initiators of the award.

Dr. Hines, the first veterinarian to receive this award, received it for outstanding contributions to public health in North Carolina, in



Dr. Martin P. Hines (left) receives the Reynolds Award from Dr. E. G. McGavran, dean, School of Public Health, University of North Carolina, and chairman of the awards committee.

adding to the body of learning in diseases of nature transmissible to man, and for constantly putting health in North Carolina above personal gain, inconvenience, and physical fatigue.

He has served as president of the National Association of State Public Health Veterinarians and, at present, he is serving as one of the vice-presidents of the AVMA.

Washington

State College of Washington Annual Awards Banquet.—The annual senior banquet of the College of Veterinary Medicine, State College of Washington, was held the evening of May



Tats Matsuoka, past-president of the Junior AVMA, receiving the Erickson Award (left). Mr. Robert Ford, executive secretary of the Washington State V.M.A., and Dr. Irving Erickson, in whose name the award was founded, are seen at the annual presentation of awards at the State College of Washington.

10, 1958, during which the following awards were made:

The H. C. Burns & Co. Award to the senior who is most proficient in small animal medicine: Jack Carkeek.

The Fred Biel Award for the student considered most proficient in small animal surgery: William Smith.

The Pfizer Award for the senior student most proficient in large animal medicine: Richard Long.

The Northwest Veterinary Supply Award to the senior student most proficient in large animal surgery: Donald Jenkins.

The Women's Auxiliary to the AVMA Award (\$50) to the senior student for outstanding activities which have contributed to the recognition of veterinary medicine and the school on campus: Donald Jenkins.

The Fred Biel Scholarship Award (\$50) to a sophomore student: Jerry LaFollette.

The Erickson Award to the third year student who has contributed the most to organizational activities in the College of Veterinary Medicine: Tats Matsuoka.

U.S. GOVERNMENT

Dr. Glascock Named Inspector in Charge of the Chicago Meat Inspection Station.—Dr. Dale W. Glascock (ISC '28), named inspector in charge of the Chicago meat inspection station of the U.S.D.A., effective June 2, 1958, succeeds Dr. O. W. Seher (COL '13) who was recently made assistant director of the meat inspection division of the Northern Area. Dr. Seher was in charge of federal meat inspection in the Chicago area for approximately ten years.

A native of Audubon, Iowa, Dr. Glascock went to Chicago from Washington, D. C., where he had been chief of the animal foods inspection division for about seven years.

STATE BOARD EXAMINATIONS

Interested persons can obtain information about applications, fees, deadlines for filing applications, and exact time and place of examinations of the respective boards by writing to the persons whose names and addresses are given below.

BRITISH COLUMBIA—November, 1958 (usually end of the second week); Vancouver. G. L. Stovell, British Columbia Veterinary Association, 3187 West 43rd Ave., Vancouver 13, secretary.

FOREIGN NEWS

Brazil

General de Pontes Examines United States' Teaching and Inspection Methods.—Brig. Gen. Eduardo de Pontes, the director of the Brazilian Veterinary Corps, recently visited Chicago, to observe the food inspection activities and training of Army veterinary officers. On a nation-wide tour of U. S. Army Medical Service facilities and installations, General de Pontes hopes that through his visit he will be able to adopt some of our methods in Brazil.



U.S. Army Medical Service

Major Clovis Gomes de Silva, assistant to Brig. Gen. de Pontes (left); Lt. Colonel W. D. Shipley, past commandant of the Meat and Dairy Hygiene School; Brig. Gen. E. de Pontes, director of the Brazilian Veterinary Corps; Colonel T. A. Ward, commandant of the Army Medical Service Meat and Dairy Hygiene School.

During the nine-day stay in Chicago, General de Pontes and his assistant, Major Clovis Gomes de Silva, toured the major food processing and packing plants, the AVMA office, and spent two days at the U. S. Army Medical Service Meat & Dairy Hygiene School, where he spoke at the student officers' graduation ceremonies and presented diplomas to the new veterinary officers.

During his trip to the AVMA, General de Pontes spoke with Dr. J. G. Hardenbergh on the inner working of the veterinary organization and profession here in the United States.

Peru

New Buildings and Facilities Under Construction for Veterinary School at San Marcos University.—The illustration shows an architectural scale model of the new buildings now being constructed at the Greater National University of San Marcos (Universidad Nacional Mayor de San Marcos) in Lima for the College of Veterinary Medicine.

First founded as an army veterinary school,

Greater National University of San Marcos, College of Veterinary Medicine
Lima, Peru

1—Administration building, library 2nd floor; 2—biochemistry—animal nutrition, physiology, pharmacology; 3—bacteriology, virus, public health, parasitology, poultry pathology; 4—auditorium; 5—general museum; 6—student center; 7—embryology, histology, anatomy, pathology, postmortem room, animal reproduction; 8—small animal hospital; 9—large animal hospital; 10—stalls; 11—wards; 12—dairy area; 13—poultry husbandry.

it was established as a part of the National University in 1946. The new facilities will cost about 10 million sols and will be completed early this fall.

Dr. Teodoro Ramos (UP '46), dean of the faculty, and Mrs. Ramos recently visited several veterinary schools in North America to observe their facilities and equipment.

COMMENCEMENTS

University of California.—At the 1958 commencement exercises of the School of Veterinary Medicine, University of California, the following 49 candidates were presented for the D.V.M. degree:

Eugene A. Adkins
William V. Allen
Myron F. Andrews
Robert P. Banbury
Charles H. Barnes
Jeri Ann Berg
Carlo Besio
Ernest W. Bizzini
Donald I. Bloomberg

John E. Brandt
Wilhelm Brannschweig
Ralph L. Cheney
Ronald J. Coggan
Kenneth W. Conklin
Ian R. Coster
Richard R. Crandall, Jr.
Alice De Groot
Clarence W. deLannoy, Jr.
Richard L. Wallis.

James M. De Vaul
Vyran E. Eberly
Kumen C. Ellsworth
Martin N. Fineman
Richard T. Flanders
Robert L. Freeman, Jr.
Leslie S. Greenbaum
Frederick A. Groverman
Theodore S. Haskell
William T. Hubbert
Larry C. Kidwell
Jerome H. Krupp
James M. Larkin
LeRoy L. LeBeuf
Wesley A. Marsden

Richard A. Mason
Richard H. McCapes
Clarence J. Mersch
Frederick W. Miller
Gerald R. Mitchell
Gary E. Montgomery
Kenneth M. Moody
James L. Naviaux
Harvey J. Olander
Samuel J. Rogers
Victor M. Shille
Gale D. Smith
Robert H. Smith
Leo P. Stringfellow
William R. Walker

(University of California commencement picture on the next page)

Graduating Class, 1958, School of Veterinary Medicine, University of California

SCHOOL

of

VETERINARY MEDICINE
University of California



Top row (left to right)—Eugene A. Adkins, William V. Allen, Myron F. Andrews, Robert P. Benbury, Charles H. Barnes, Jeri Ann Berg.

Second row—Carlo P. Besio, Ernest W. Bizzini, Donald I. Blomberg, John E. Brandt, Wilhelm Brennschweig, Ralph L. Cheney, Ronald J. Coggan.

Third row—Kenneth W. Conklin, Ian R. Coster, Richard R. Crandall, Jr., Alice A. De Groot, Clarence W. de Lannoy, Jr., James M. De Vaul, Vyran E. Eberly, Kumen C. Ellsworth.

Fourth row—Martin N. Fineman, Richard T. Flanders, Robert L. Freeman, Jr., Leslie S. Greenbaum, Dean D. E. Jasper, Frederick A. Groverman, Theodore S. Haskell, William T. Hubbert, Larry C. Kidwell.

Fifth row—Jerome H. Krupp, James M. Larkin, LeRoy L. LeBeuf, Wesley A. Marsden, Richard A. Mason, Richard H. McCapes, Clarence J. Mersch, Frederick W. Miller, Gerald R. Mitchell, Gary E. Montgomery.

Sixth row—Kenneth M. Moody, James L. Naviaux, Harvey J. Olander, Samuel J. Rogers, Victor M. Shille, Gale D. Smith, Robert H. Smith, Leo P. Stringfellow, William R. Walker, Richard L. Wallis.

Graduating Class, 1958, School of Veterinary Medicine, Kansas State College



Top row (left to right)—Patricia A. Cole, G. A. Roggendorff, W. E. Birtell, R. D. Johansen, W. H. Bales, Jr., J. W. Judy, Jr., G. D. Rousseau, D. L. F. Pohiman, K. D. Weide, K. D. Beeman, L. D. Kester.

Second row—W. A. Schleifer, J. B. Shields, O. G. Post, I. F. Rodman, Jr., P. R. Randall, J. M. Woods.

Third row—R. A. Arnold, D. E. Baldwin, R. F. Sand, T. J. Landis, W. E. Ketter, J. W. Johnson, E. M. Coon, D. F. Jarchow, N. M. Held, D. L. Bokelman, R. M. Grossdier, K. C. Sherman.

Fourth row—F. R. Robinson, G. P. Looby, H. E. Brune, G. A. Clarke, R. H. Pierson, II, M. L. Sutton, R. M. Hodgson, J. R. Osterheld, J. H. Sherrod, R. B. Talbot, H. R. Sinclair, D. D. Duntz.

Fifth row—R. L. Cleveland, A. E. Wesley, E. L. Chatfield, J. D. Smith, W. W. Ogborn, D. L. Madden, W. D. Nichols, L. R. Parton, J. L. Murray, D. D. Sharp, K. R. Rhoades, R. L. Ganoung.

Sixth row—J. E. Watt, D. W. Fitzgerald, G. W. Olson, H. J. Kugler, N. J. Sojka, E. E. Tobler, E. B. York, C. E. Sevy, J. V. Krone, W. B. Wren, W. E. Schaulis, E. H. Strevell.

Kansas State College.—At the 1958 commencement exercises of the School of Veterinary Medicine, Kansas State College, the following 65 candidates were presented for the D.V.M. degree:

Richard A. Arnold
Donald E. Baldwin
William H. Bales, Jr.
Keith Beeman
William E. Birtell
Delwin L. Bokelman
Harold E. Brune
Elton L. Chatfield
G. A. Clarke
Robert Cleveland
Patricia A. Cole
Everett M. Coon
Delvin D. Duntz

Don Wayne Fitzgerald
Raymond Larry Ganoung
Ralph Grossdier
Norman M. Held
Robert M. Hodgson
Donald F. Jarchow
Richard D. Johansen
John W. Johnson
John W. Judy, Jr.
Larry D. Kester
William E. Ketter
James V. Krone
H. John Kugler
Edward B. York

Thomas J. Landis
George P. Looby
David L. Madden
James E. Murray
Warren D. Nichols
William Ogborn
George W. Olson
John R. Osterheld
Lawrence R. Parton
Robert H. Pierson, II
Don Pohiman
Otis G. Post
Philip R. Randall
Keith Ray Rhoades
Farrel R. Robinson
Ivan F. Rodman, Jr.
George A. Roggendorff
G. D. Rousseau
Robert F. Sand
Robert F. Sand
Edward B. York

William F. Schaulis
Wilbur A. Schleifer
Galvin E. Sevy
Darrell D. Sharp
Keith C. Sherman
James H. Sherrod
James B. Shields
Hal R. Sinclair
James D. Smith
Nickolas J. Sojka
Edward H. Strevell
Max "L." Sutton
Richard B. Talbot
Earl E. Tobler
John Watt
Kenneth D. Weide
Albert E. Wesley
John M. Woods
Wallace B. Wren

DEATHS

Star Indicates member of AVMA

★**Nelson S. Mayo** (CVC '89), twice secretary of the AVMA, long time member of its Committee (later Council) on Education and an honor roll member of the Association since 1941, died on July 5, 1958, at his house in Highland Park, Ill., at the age of 91.



Dr. Nelson S. Mayo

Born in Marshall, Mich., Nov. 16, 1866, Dr. Mayo attended local schools before entering Michigan State College where he received the B.S. degree in 1888. The following year he graduated from the Chicago Veterinary College and then returned to Michigan State to earn an M.S. degree in 1890. During part of this time, he was assistant veterinarian in the state agricultural experiment station. From 1890 to 1897, he was professor of veterinary science at Kansas State Agricultural College and then held the same position at Connecticut Agricultural College for four years, before returning to Kansas State to head its veterinary science work again. While teaching, he wrote a book for agricultural students on animal diseases.

In 1904, Dr. Mayo was appointed vice-director and chief of the department of animal industry at the agricultural experiment station, Republic of Cuba, and helped establish the College of Veterinary Medicine at the University of Havana in 1907. For this and other accomplishments in building their veterinary services, Cuba honored Dr. Mayo in 1942 by naming the new nutritional laboratory building at the University for him.

In 1909, Dr. Mayo returned to the States and was professor of animal husbandry and veterinary science at Virginia Polytechnic Institute until 1913. He then became manager of the veterinary department at Abbott Laboratories, a position he held until he retired a number of years ago.

Always active in AVMA affairs, he not only served two terms as secretary (1913-1915 and 1918-1922) but also on its educational committees. He was a member of the former Committee on Intelligence and Education from 1915 to 1917 (chairman in 1916) and of its successor, the Committee on Education, from 1928 to 1943, being chairman for the last 13 of those years. It was during this period that classification of veterinary colleges under the Committee's jurisdiction was approved in principle by the Association in 1933. But, when the Committee presented its classification of the schools in its 1934 report, this section of the report was not adopted by the House of Representatives.

Under Dr. Mayo's leadership, the Committee drafted the first "Essentials of An Accepted Veterinary School", a document which, in revised form, has guided the American colleges ever since, in meeting AVMA standards for accreditation. Dr. Mayo also played a key role in the Association's decision to publish its own journal. While secretary in 1915, he made the recommendation that led to the acquisition of the *American Veterinary Review* and its rebirth as the JOURNAL of the AVMA. He also edited the first *AVMA Directory* which was issued while he was secretary in 1920-1921.

Surviving are a son, Robert S. Mayo of Lancaster, Pa.; two daughters, Mrs. Marguerite Lockhart, widow of the late Dr. Ashe Lockhart, of Kansas City, Mo., and Mrs. Mary Freytag of Lake Forest, Ill.

Interment was in Evergreen Cemetery, Lake Orion, Mich., on July 7.

• • •

★**Albert Hjärre** (STK '22), 61, director of the State Veterinary Medical Institute, Stockholm, Sweden, noted animal pathologist and honorary member of the AVMA, died on April 22, 1958, after a short illness.

Born at Stilby, Sweden, on Nov. 23, 1897, Dr. Hjärre received his pre-veterinary education at the University in Lund in 1915-1916 and then attended the Royal Veterinary College in Stockholm from 1916 to 1922. Following graduation, he was first an assistant professor, then associate and, from 1930 to 1944, full professor in the institute of anatomy and pathology. In 1944, he was appointed director of the State Veterinary Medical Institute, which had a staff of nearly 200, for research and diagnosis of animal diseases.

Dr. Hjärre's work on epizootic diseases included infectious equine anemia, tuberculosis in dogs and cats, swine influenza, virus pneumonia, pasteurellosis in wild animals, and bovine leukosis. He was a member of the advisory board of "Advances in Veterinary Science" and a member of the editorial staffs of the *Zentralblatt für Veterinärmedizin* and *Nordisk Veterinärmedicin*.

He received numerous honors and awards in-

cluding: Dr. Med. h.c., from the Stockholm Medical Academy; the same degree from the University of Utrecht; and the D.V.M. degree from the Free University of Berlin. He was an honorary member of the AVMA (1949) and of the Sociedad Veterinaria of Madrid. He was active in international veterinary affairs in which he always tried to achieve cooperation and was well known to a large number of veterinary scientists in this country through his visits to the United States.

Clinton E. Lucas (CVC '09), 77, Olney, Ill., died at the Richland Memorial Hospital, where he had been a patient for ten days on May 16, 1958.

Dr. Lucas had practiced in Olney since 1909. He was vice-president and director of the First National Bank in Olney, a member of the A.F. & A. Masons, Order of the Eastern Star, and of the Elks Club.

He had been active in Richland County Fair work, serving as president and director of the Fair board for several years. He is survived by his widow, Lottie West Lucas, one brother, and two sisters.

William H. Marriott (ONT '07), 75, Union, Ont., died in the St. Thomas Elgin General Hospital on May 11, 1958.

Albert L. Metz, (SF '12), 75, Oxnard, Calif., died at his home May 27, 1958, after a lingering illness.

Dr. Metz was born in Ohio in 1883, but had resided in the Oxnard area since 1918 where he remained active in the veterinary profession up until the time of his retirement in 1955.

He was a member of the Ventura County Sheriff's Posse and of the Oxnard Elks Club. He is survived by a niece and a nephew.

★**James H. Moret** (ISC '42), 42, Morrison, Ill., died on June 9, 1958, after an illness of one year.

Born in 1915 at Boyden, Iowa, Dr. Moret received his pre-veterinary training at the University of Iowa before enrolling at Iowa State College. Following graduation, he practiced at Leroy, Ill., for three years before joining Dr. A. M. Olson in the Morrison Veterinary Clinic.

Dr. Moret was a member of the AVMA (joined 1942), the Illinois State and Northern Illinois veterinary medical associations, the Reformed Church, and the Chamber of Commerce.

Surviving are his widow, the former Irene LeFever of Bloomington, Ill., whom he married in 1943, two daughters, and a son; also his father, stepmother, two sisters, and three brothers.

George F. Nixon, Sr., (OSU '36), 52, Alliance, Ohio, died of a cerebral hemorrhage at his home in Ontario, Can., on May 9, 1958.

For the last four years, Dr. Nixon had been associated with the Alliance Animal Hospital. He was a member of the Kiwanis Club and the Masonic Lodge of Sullivan, Ohio. He was a past-president of the Stark County V.M.A.

Dr. Nixon is survived by his wife, Ida, a daughter, and one son, George F., Jr., (OSU '54), also a veterinarian.

★**Kenneth J. Thomas** (UP '36), 45, Johnstown, Pa., died May 5, 1958, in Memorial Hospital, a few days before his 46th birthday.

Dr. Johnson had practiced in the Johnstown area for nearly 20 years. Active in civic affairs, he was a member of Southmont Borough Council and of the borough's recreation commission.



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Women's Auxiliary Meeting in Philadelphia.—The forty-first annual meeting of the Women's Auxiliary to the AVMA will be held in Philadelphia, August 16-21, during the ninety-fifth annual meeting of the AVMA. The president, Mrs. L. H. Moe, and her officers cordially invite all women attending the convention to attend the House of Representative meetings on Tuesday, August 19, and the auxiliary workers' conference on Wednesday, August 20.

The Local Committee in Philadelphia suggests that light-weight summer clothes will be suitable, with a sweater in the background for the air-conditioned meeting rooms.

APPLICATIONS

Applicants—Members of Constituent Associations

In accordance with paragraph (e) of Section 2, Article X, of the Administrative Bylaws, the names of applicants residing within the jurisdictional limits of a constituent association shall be published once in the JOURNAL.

The following applicants have been certified by the secretary of the applicable constituent association in accordance with paragraph (c) Section 2, Article X, of the Administrative Bylaws.

BAKER, BERNARD M., Hwy 105 E., East Prairie, Mo., D.V.M., University of Missouri, 1954.

BLACKBURN, EARL G., Rt. 2, Quakertown, Pa., V.M.D., University of Pennsylvania, 1953.

DOWNING, CHARLES W., 421 S. Garfield St., Lyons, Kans., D.V.M., Kansas State College, 1950.

GURSS, GERALD D., Lyndon, Kan., D.V.M., Kansas State College, 1943.

HUGHES, CLINTON P., 2330 Mt. Vernon Rd., S.W., Roanoke, Va., D.V.M., Alabama Polytechnic Institute, 1943.

JOHNSON, EDGAR S., 1638 South B. Street, Elwood, Ind., D.V.M., Ohio State University, 1935.

LESEHORA, ANTHONY, 1137 N. E. 9th Ave., Ft. Lauderdale, Fla., V.M.D., University of Pennsylvania, 1956.

NEEDHAM, THOMAS C., 3603 Wrightsville Ave., Wilmington, N. Car., D.V.M., Alabama Polytechnic Institute, 1952.

Applicants—Not Members of Constituent Associations

In accordance with paragraph (e) of Section 2, Article X, of the Administrative Bylaws, the names of applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months.

The first notice shall give the applicant's full name, school, and year of graduation, post office address, and the names of his endorsers.

First Listing

SCOTT, LYLE, Jr.

81st Medical Detachment (VFI) APO 258, New York, N. Y.

D.V.M., Iowa State College, 1945.

Vouchers: C. W. Betzold and W. F. Collins.

Second Listing

BOND, GEORGE H., D.V.M. 421-C Washington Blvd., Presidio of San Francisco, Calif.

RAULSTON, GILBERT L., D.V.M. Chemical Warfare Laboratory Building 355, Army Chemical Center, Md.

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

First Listing

A. & M. College of Texas

GREZAFFI, ANTHONY J., D.V.M. 1001 Parent St., New Roads, La.

Vouchers: J. N. Chastain and R. J. Beamer.

PATRICK, VERGIL, D.V.M. 408 B. 2nd St., College Station, Texas.

Vouchers: A. A. Lenert and W. M. Romane.

ROSS, TOM W., D.V.M. Star Rt., Manchester, Tenn.

Vouchers: G. S. Trevino and J. P. Davis.

WYATT, RICHARD T., D.V.M. 4521 Ave. C., Austin, Texas.

Vouchers: J. P. Davis, Jr. and G. S. Trevino.

Colorado State University

ALLEN, ROBERT E., D.V.M. 425 S. Forest St., Denver, Colo.

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BAILEY, ROBERT B., D.V.M. 248 N. W. Garden Valley Blvd., Roseburg, Ore.

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BOHLENDER, ROBERT E., D.V.M. 309 Locust St., Fort Collins, Colo.

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BOYD, LAURENCE J., D.V.M. 7339 N. 27th Ave., Phoenix, Ariz.

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CADY, ALONZO E., Jr., D.V.M. 115 S. Sherwood St., Fort Collins, Colo.

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CHRISTIANSEN, DALE, D.V.M. 462 N. 2nd E., Logan, Utah.

Vouchers: W. D. Carlson and L. K. Wayt.

ELLSWORTH, JAY P., D.V.M. 1518 S. Corona St., Denver, Colo.

Vouchers: G. Cholas and J. T. Collins.

ETTER, MILTON W., D.V.M. Animal Clinic, Boulder, Colo.

Vouchers: J. R. Popish and L. C. Moss.

FALLOON, RICHARD A., Jr., D.V.M. La Porte, Colo.

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FARMER, DARRELL D., D.V.M. 1305 S. 2nd St., Tucumcari, N. M.

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FERGUSON, BEN R., Jr., D.V.M. 317 W. Mulberry St., Fort Collins, Colo.

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GERNER, CHARLES L., D.V.M. c/o L. E. Hansen, Vaughn, Mont.

Vouchers: J. R. Popish and L. K. Wayt.

HAM, DONALD J., D.V.M. Glendive, Mont.

Vouchers: G. Cholas and W. A. Aanes.

HANCOCK, WILLIAM F., D.V.M. 214 E. 11th St., Wahoo, Neb.

Vouchers: W. A. Aanes and G. Cholas.

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1. Harris, J.R., and Clarkson, T.B., Prevention of Relapses in Milk Fever, *Vet. Medicine*, 12:696 (Dec. 1955)

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COMING MEETINGS

Connecticut Veterinary Medical Association. Annual outing meeting. Rosewood Inn and Grove, Simsbury, Conn., Aug. 6, 1958. E. H. Patchen, 289 New Haven Ave., Milford, Conn., secretary.

College of Veterinary Surgeons of Province of Quebec. Annual meeting, Quebec, Aug. 8-9, 1958. Jacques Saint Georges, St. Hyacinth, Que., public relations committee.

Mississippi State Veterinary Medical Association. Fifty-second annual meeting. Buena Vista Hotel, Biloxi, Aug. 10-12, 1958. H. F. McCrory, State College, Miss., secretary.

American Association of Veterinary Bacteriologists. Annual meeting. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Aug. 16, 1958. C. H. Cunningham, Michigan State University, College of Veterinary Medicine, East Lansing, secretary.

American Veterinary Medical Association. Ninety-Fifth Annual Meeting. Convention Hall, Philadelphia, Pa., Aug. 18-21, 1958. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

Louisiana Veterinary Medical Association, Inc. Annual meeting. Montelone Hotel, New Orleans, Aug. 24-26, 1958. R. R. Gibbs, 6421 Chef Menteur Highway, New Orleans, La., program chairman.

New York State Veterinary Medical Society. Sixty-seventh annual meeting. Concord Hotel, Kiamaha Lake, N. Y., Sept. 4-6, 1958. Miss Joan S. Halat, 803 Varick St., Utica, N. Y., executive secretary.

New Mexico Veterinary Association. Annual meeting. La Posada Hotel, Santa Fe, Sept. 8-9, 1958. E. R. Leslie, 920 N. Main, Carlsbad, N. M., secretary.

Washington State Veterinary Medical Association. Annual meeting. Hotel Olympian, Olympia, Sept. 8-10, 1958. F. M. Shigley, P.O. Box 1163, Olympia, Wash., program chairman.

South Dakota Veterinary Association. Meeting. Hotel Cateract, Sioux Falls, Sept. 16-17, 1958. G. E. Duncan, Tyndall, secretary.

New England Veterinary Medical Association. Annual meeting. Hotel Wentworth-By-The-Sea, Portsmouth, N. H., Sept. 28-Oct. 1, 1958. C. Lawrence Blakely, 180 Longwood Ave., Boston 15, Mass., secretary-treasurer.

Missouri, University of. Annual short course for graduate veterinarians. School of Veterinary Medicine, Columbia, Mo., Oct. 6-7, 1958. Cecil Elder, chairman.

Florida State Veterinary Medical Association. Annual meeting. Galt Ocean Mile Hotel, Fort Lauderdale, Oct. 12-14, 1958. A. R. Chambers, 6116 Main St., Jacksonville, secretary.

District of Columbia Veterinary Medical Association. Annual all-day meeting. Walter Reed Army Medical Center, Washington, D.C., Oct. 14, 1958. W. I. Gay, 5200 Chandler St., Bethesda, Md., secretary-treasurer.

Eastern Iowa Veterinary Association, Inc. Annual meeting. Hotel Roosevelt, Cedar Rapids, Oct. 16-17, 1958. F. E. Brutsman, Traer, Iowa, secretary-treasurer.

Texas Veterinary Medical Association. Annual meeting. Stephen F. Austin Hotel, Austin, Oct. 19-21, 1958. Paul B. Blunt, 710 Maverick Bldg., San Antonio, secretary.

Southern Veterinary Medical Association. Annual meeting. Claridge Hotel, Memphis, Tenn., Oct. 26-30, 1958. A. A. Husman, P.O. Box 91, Raleigh, N. C., secretary.

Arizona Veterinary Medical Association. Annual meeting. Yuma, Ariz., Dec. 7-9, 1958. R. E. McComb, Jr., 4730 N. 7th Ave., Phoenix, program chairman.

Tennessee Veterinary Medical Association. Annual meeting. Noel Hotel, Nashville, Jan. 11-13, 1959. H. W. Hayes, 5009 Clinton Pike, Knoxville, secretary-treasurer.

Oklahoma Veterinary Medical Association. Annual meeting. Mayo Hotel, Tulsa, Jan. 25-27, 1959. M. N. Riemenschneider, 122 State Capitol Bldg., Oklahoma City, secretary.

Ohio State Veterinary Medical Association. Annual convention. Neil House Hotel, Columbus, Feb. 4-6, 1959. Harry C. Sharp, 1411 W. Third Ave., Columbus, Ohio, executive secretary.

Applications—continued from p. 35

SINCLAIR, DUNCAN G., D.V.M., Ontario Veterinary College, Guelph, Ont.

WALCROFT, MICHAEL J., D.V.M., Kingston, N. S.

University of Pennsylvania

BEHLER, WILEY V., V.M.D., 815 E. Montgomery Ave., Narberth, Pa.

DURFEE, GEORGE F., V.M.D., Box 25, N. Hackensack, N. J.

FLICKINGER, GEORGE L., Jr., V.M.D., 4002 Pine St., Philadelphia, Pa.

HOPWOOD, RONALD T., V.M.D., 1110 N. W. Little River Dr., Miami, Fla.

McKISSICK, GAYLORD E., V.M.D., 519 S. 45th St., Philadelphia, Pa.

MAEL, MORTON S., V.M.D., Village St., Millis, Mass.

TIBOLLA, BRUNO J., Jr., V.M.D., Oak Ave., Rt. 2, Blackwood, N. J.

TODD, GLEN C., V.M.D., Rt. 4, Crawfordsville, Ind.

TRAVIS, STANLEY O., V.M.D., Rt. 1, Troy Rd., Keene, N. H.

WALTERS, SHIRLEY R., V.M.D., 4418 Pine St., Philadelphia, Pa.

WEBBER, NATALIE K., V.M.D., 20 Adams Ave., Everett, Mass.

Sixth International Congresses on Tropical Medicine and Malaria. Lisbon, Portugal, Sept. 5-13, 1958. Professor Manuel R. Pinto, Institute of Tropical Medicine, Lisbon, secretary-general. (Membership application forms may be obtained by U.S. veterinarians by writing to the AVMA.)

Veterinary Congress of Brussels. Brussels, Belgium, Sept. 6-17, 1958. Further information available from Bureau, 35 Rue de la Brasserie, Brussels 5.

German Veterinary Association. Regular biennial meeting. Hannover, Germany, Sept. 19-21, 1958. Dr. Karl Ohly, 123 Forsthausstrasse, Frankfurt/Main, Germany, president.

Seventh International Congress on Hydatid Disease. Beirut, Lebanon, Sept. 20-22, 1958. Dr. Elias Sader, c/o Lebanese Order of Physicians, P.O.B. 640, Beirut, Lebanon, secretary-general. (Applications to present papers must be accompanied by an abstract.)

International Veterinary Congress. Sixteenth session. Madrid, Spain, May 21-27, 1959. Prof. Pedro Carda A., general secretary, Calle Villanueva 11, Madrid.

U.S. COMMITTEE: Dr. W. A. Hagan, chairman, New York State Veterinary College, Ithaca, N. Y.; Dr. J. G. Hardenbergh, secretary, 600 S. Michigan Ave., Chicago 5, Ill.

Third World Congress on Fertility and Sterility. Amsterdam, Holland, June 7-13, 1959. Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland, honorary secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. Dr. G. W. Jones, Main St., Prattville, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the second Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, Decatur, Ala., secretary.

North East Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16 St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleus, 2335 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. James L. Frederickson, 17 Niles St., Bakersfield, Calif., secretary-treasurer.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643-N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. M. Granfield, 2600 W. El Camino Real, San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert L. Chandler, P.O. Box 8, Ukiah, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Dr. V. H. Austin, 14931 Oxford St., Van Nuys, secretary-treasurer.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. Dr. Rolf Reese, 23815 Ventura Blvd., Calabasas, Calif., secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinary Medical Association, the second Thursday of each month. D. E. Britten, 544 N. Ben Maddox, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

ANIMAGRAPH

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FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. William F. Casler, 2540 30th Ave., N., St. Petersburg, secretary-treasurer.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at #10 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4140 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3002 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

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GEORGIA—Atlanta Veterinary Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Donald C. Ford, Forest Park, secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. Alfred G. Schiller, Veterinary Clinic, University of Illinois, Urbana, secretary-treasurer.

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Dr. Dvorkovitz Named President of Jensen-Salsbury Laboratories

Dr. Vladimir Dvorkovitz became president and general manager of Jensen-Salsbury Laboratories, Inc., Kansas City, June 9, 1958, according to H. S. Richardson, Jr., president of Vick Chemical Company. Dr. Dvorkovitz succeeds Charles M. McCallister who became president and general manager of the National Drug Company, Philadelphia.

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Dr. Vladimir Dvorkovitz

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Dr. Dvorkovitz joined Jensen-Salsbury in 1955 as assistant general manager and, in 1956, became vice-president in charge of research and production. A graduate of the University of London, the new president's prior experience includes posts as chief chemist and director of laboratories of the Diversy Corporation, Chicago; and as research chemist with Sharples Chemicals, Wyandotte, Mich., and Burroughs-Wellcome & Company, Tuckahoe, N. Y.

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INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. Dr. J. G. Irwin, Iowa City, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer, Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. Richard Baum, Osage, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirell, Versailles Rd., Frankford, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday of each month in Louisville or within a radius of 50 miles, except January, May, and July. G. R. Comfort, 2102 Reynolds Lane, Louisville, Ky., secretary-treasurer.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Norman Herbert, 3506 Joann Drive, Baltimore 7, Md., secretary.

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REFERENCES: 1. Hesler, J. E.: *Vet. M.* 90:605 (Nov.) 1955.
2. Belloff, G. B.: *Calif. Vet.* 9:27 (Sept.-Oct.) 1956.
3. Pollock, S.: *J. Am. Vet. M. Ass.* 129:274 (Sept.) 1956.

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Make your diagnosis from the picture below—then turn the page ▶



Figure 1

History.—A mature, black swan had been lame in the right leg for at least two weeks. The bird otherwise was healthy. A radiograph, ventrodorsal view, was taken (fig. 1).

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—A well-defined, fine granular area of opaqueness was visible in the region anterior to the right acetabulum and proximal portion of the shaft of the femur. A tumor, possibly an osteogenic sarcoma, was suspected. Another radiograph, a lateral view (fig. 2, arrow), showed the opaque object to be in the ventral portion of the abdominal cavity. This was finally determined to be ground limestones in the gizzard.



Fig. 2—Radiograph, lateral view, showing the opaqueness to be in the ventral portion of the abdominal cavity of the swan.

This case was submitted by Dr. Jack Haggard, Yarborough Clinic, Miami, Fla.

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ciation, the first Wednesday of every month at 7 p.m. Frank A. Carter, P.O. Box 78, Carson City, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Frank A. O'Donnell, Parkville, Mo., secretary-treasurer.

NEVADA—Western Nevada Veterinary Society, the first Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Highstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91

Lincoln Park South, Newark, N. J. Myron S. Aslein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. Burritt Lupton, 369 Franklin Ave., Wyckoff, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. R. M. Sauer, secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, third Wednesday of each month, Fez Club, Albuquerque, N.M. Jack Ambrose, 3018 N. Rio Grande Blvd., Albuquerque, secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College, Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. T. L. James, Box 243, Newton, N. Car., secretary.

Twins Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leek St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Asheville, N. Car. Vilu Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuler's Wigwam, 6210 Hamilton Ave., at North Bend Road. G. C. Lewis, Cincinnati, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. D. J. Kern, Killbuck, Ohio, secretary-treasurer.

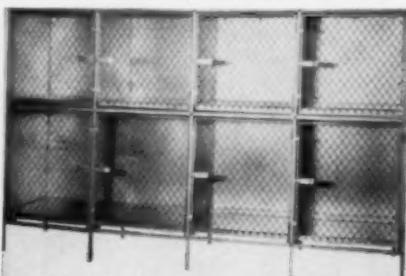
Mahoning County Veterinary Medical Association, the third Tuesday of each month, at 9:00 p.m., Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

Miami Valley Veterinary Medical Association, the first

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Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvans, Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Lohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m., Ireland's Restaurant, Lloyds', 718 N.E. 12th Ave., Portland. Donald L. Moyer, #415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania, School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blackburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association,

the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. I. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowa (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 9th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). D. F. Ludvigson, Ridgeland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

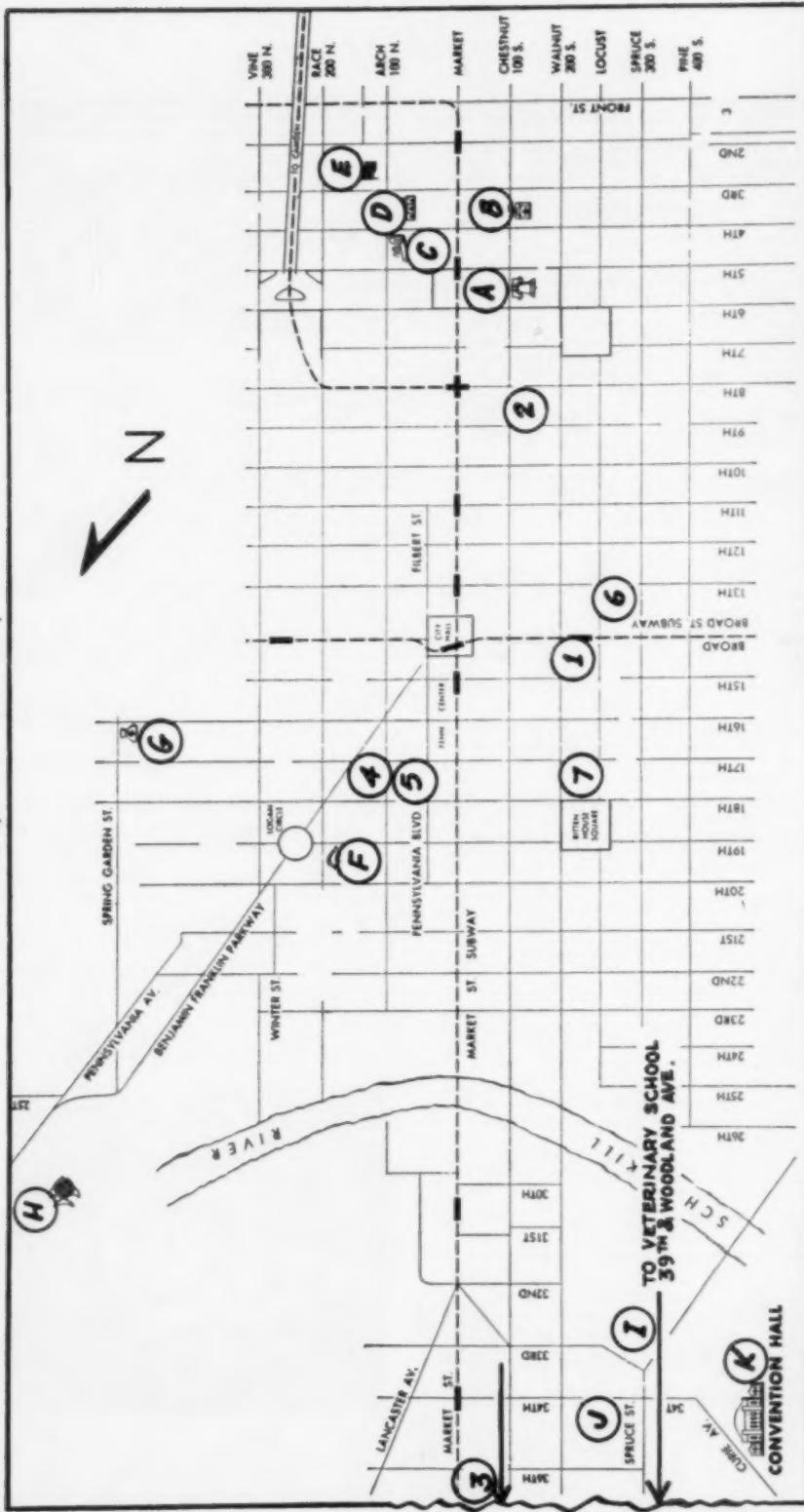
Erysipelas in a Hyena.—A striped hyena in a zoo, in Poland, died of subacute erysipelas infection. The diagnosis was confirmed bacteriologically.—*Med. Wtrryn.* (April, 1957): 577.

When Columbus discovered the New World, the only domestic animals in North America were turkeys and dogs—*All About Dogs, May, 1958.*

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Philadelphia Location Map



1. Bellevue-Stratford Hotel
 2. Benjamin Franklin Hotel
 3. Penn-Sherwood
 4. Robert Morris
 5. Sheraton Hotel
 6. Sylvania Hotel
 7. Warwick Hotel
 A. Independence Hall
 B. Carpenters' Hall
 C. Franklin's Grave
 D. Friends' Meeting House
 E. Betsy Ross House
 F. Academy of Natural Sciences
 G. U.S. Mint (Guided Tours)
 H. Aquarium
 I. University Museum
 J. University of Pennsylvania
 K. Convention Hall

HOTEL RESERVATIONS — PHILADELPHIA CONVENTION

Ninety-Fifth Annual AVMA Meeting, Aug. 18-21, 1958

All requests for hotel accommodations will be handled by a Housing Bureau in cooperation with the Committee on Local Arrangements. The Bureau will clear all requests and confirm reservations.

Hotels and Rate Schedule

Hotel	Single	Double	Twin	Suite
1. Bellevue-Stratford †	\$ 9.00-11.00	\$12.00-16.00	\$12.00-17.00	\$30.00-50.00*
2. Benjamin-Franklin †	9.00-11.00	12.00-15.00	16.00-18.00
3. Penn Sherwood †	6.00- 7.50	11.00	13.50-14.00	15.50*
4. Robert Morris †	6.00	9.50	11.00
5. Sheraton †	9.85-13.50	13.00	15.00-17.00	30.50-41.00*
6. Sylvania ††	7.50- 9.50	10.00-13.00	25.00-27.50*
7. Warwick †	10.00-12.00	15.00-18.00	30.00-35.00*

† Air-Conditioned

†† Partly Air-Conditioned

* 2-room suite

FAMILY PLAN — The 7 hotels listed above offer a "Family Plan" whereby children under 14 years of age receive accommodations free of charge. For more detailed information, contact the Housing Bureau.

—Tear Off—

RESERVATION FORM — AVMA CONVENTION — PHILADELPHIA

To: Housing Bureau, Philadelphia Convention and Visitors Bureau, Inc., Penn Square Building, Juniper & Filbert Sts., Philadelphia, Pa.

Hotel	Accommodations
(Three choices MUST be shown)	Single Room(s) @ \$ _____
First choice hotel _____	Double Room(s) @ \$ _____
Second choice hotel _____	Twin-bed Room(s) @ \$ _____
Third choice hotel _____	2-Room Suite @ \$ _____

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() p.m.

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DEADLINES

1st of month issue — 8th of month preceding date of issue.

15th of month issue — 22nd of month preceding date of issue.

Names of classified advertisers using key letters can not be supplied. Address your reply to the box number, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be sent to the advertiser.

Wanted—Veterinarians

Ambitious veterinarian wanted to operate small animal hospital in District of Columbia; salary plus percentage; apartment available. Address "Box K 16," c/o JOURNAL of the AVMA.

Wanted—veterinarian to operate two-year-old animal hospital, Phoenix. Percentage; lease or purchase basis. Good future for right man. Write Dr. Shapera, 4122 N. 36th St., Phoenix, Ariz.

Maryland—licensed, capable small animal man, \$125 per week starting salary. In letter, state pertinent facts such as age, ability, experience, availability. Bonus if deserving. Address "Box M 4," c/o JOURNAL of the AVMA.

Wanted—veterinarian to run new small animal hospital, California. Major percentage of net, excellent opportunity for permanent position, prefer recent graduate. Address "Box M 8," c/o JOURNAL of the AVMA.

Veterinarian wanted, mixed practice. State qualifications, references, salary expected. Must have Florida license. Sale or lease in few months if satisfactory. Address "Box M 12," c/o JOURNAL of the AVMA.

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Wanted—assistant veterinarian for small animal practice only. Arizona license preferred. With living quarters, separate three-bedroom house. Address "Box M 14," c/o JOURNAL of the AVMA.

Veterinarian to assist in 70 per cent small and 30 per cent large animal practice. We have a new, completely-equipped hospital located in a large southern city, and an established practice. Excellent opportunities for the right person. Address "Box M 18," c/o JOURNAL of the AVMA.

Wanted—Positions

Veterinarian, age 30, with small animal experience, desires association with small animal practice in South or Southeast leading to partnership or purchase. Excellent references. Address "Box L 33," c/o JOURNAL of the AVMA.

Graduate, 1955, being discharged from service in October, desires position with small animal practitioner. Eighteen months' small animal experience prior to service. Address "Box M 7," c/o JOURNAL of the AVMA.

Graduate (1955) desires position or partnership in active small animal hospital in Texas, Oklahoma, or Arkansas. Three years' experience in medicine, surgery, and hospital management. Married with children, military service completed, 29 years old. Have some capital and complete hospital equipment

if partnership is arranged. Am more interested in salary than short working hours. Address "Box M 9," c/o JOURNAL of the AVMA.

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Wanted—Practices

Florida—to purchase or be assistant with future ownership small animal hospital pending state exam. Answer all replies. Address "Box L 1," c/o JOURNAL of the AVMA.

Veterinarian with 7 years' general practice experience interested in association leading to partnership or purchase of general practice in southern or central Minnesota. Married, children. Address "Box M 13," c/o JOURNAL of the AVMA.

For Sale or Lease—Practices

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Established mixed practice in western Nevada for sale. Modern home, attached hospital, excellent climate. \$4000 down, balance terms. Dr. M. Houston, Yerington, Nev.

Long-established small animal hospital for sale, large city northern Ohio. Gross \$40,000. No real estate. \$20,000. Address "Box L 20," c/o JOURNAL of the AVMA.

Small animal hospital with bedroom, central Connecticut. Equipment one year old. Excellent opportunity for young veterinarian. Address "Box L 13," c/o JOURNAL of the AVMA.

Established practice for sale in north central New England. A prosperous general practice. Accent on dairy cattle, with sufficient light horse and pet work to make fine combination. Sale price \$37,000, includes valuable real estate and equipment. Address "Box L 21," c/o JOURNAL of the AVMA.

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Wanted—one copy, Am. J. Vet. Res., October, 1954, and copies or volumes prior to 1954. Also J.A.V.M.A. prior to July, 1948. Write Donald E. Davis, Londres 40, Mexico 6, D.F., Mexico.

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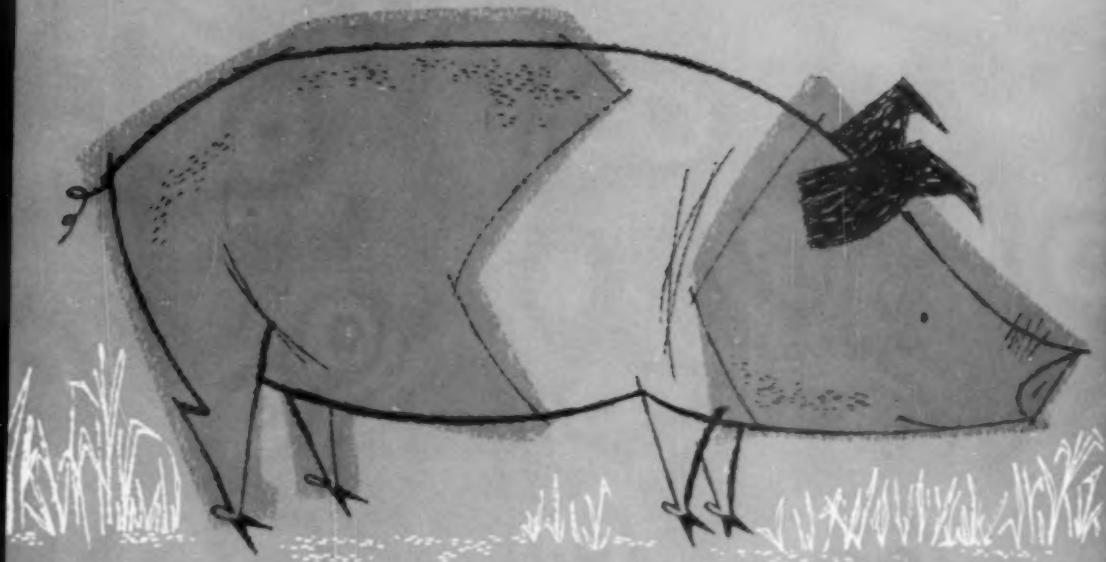
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